SERUM LIPID PROFILE – A PREDICTOR OF PRE-ECLAMPSIA IN PIH MOTHER COMPARATIVE STUDY

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

DOCTOR OF OBSTETRICS AND GYNAECOLOGY

(Branch II) M. S. (OBSTETRICS AND GYNAECOLOGY)

of

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CHENNAI- 600032

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 11

MAY 2019

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"SERUM LIPID PROFILE – A PREDICTOR OF PRE-ECLAMPSIA IN PIH MOTHER COMPARATIVE STUDY"** submitted by **Dr.Shanmuga Priya.M** to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S. Degree Branch – II (Obstetrics and Gynaecology) is a bonafide research work carried out by her under direct supervision & guidance.

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CERTIFICATE

This is to certify that the Dissertation **"SERUM LIPID PROFILE – A PREDICTOR OF PRE-ECLAMPSIA IN PIH MOTHER COMPARATIVE STUDY"** presented here in by **Dr.Shanmuga Priya.M** is an original work done in the Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.S. (Branch II) Obstetrics and Gynaecology under my guidance and supervision during the academic period of 2016 -2019.

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DECLARATION

I solemnly declare that the dissertation titled **"SERUM LIPID PROFILE – A PREDICTOR OF PRE-ECLAMPSIA IN PIH MOTHER COMPARATIVE STUDY"** is done by me at Tirunelveli Medical College hospital, Tirunelveli. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, or diploma to any other University, Board, either in or abroad.

The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.S. Degree (Branch II) in Obstetrics and Gynaecology.

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CERTIFICATE – II

This is to certify that this dissertation work title **"SERUM LIPID PROFILE – A PREDICTOR OF PRE-ECLAMPSIA IN PIH MOTHER COMPARATIVE STUDY"** of the candidate **Dr.Shanmuga Priya.M** with registration Number **221616351** for the award of **M.S.** Degree in the branch of **OBSTETRICS AND GYNAECOLOGY (II).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **0 percentage** of plagiarism in the dissertation.

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PROTOCOL TITLE: SERUM LIPID PROFILE – A PREDICTOR OF PRE-ECLAMPSIA IN PIH MOTHER – COMPARATIVE STUDY
PRINCIPAL INVESTIGATOR: POST GRADUATE STUDENT
DESIGNATION OF PRINCIPAL INVESTIGATOR: DR.M.SHANMUGA PRIYA, MBBS.,
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear Dr.M.SHANMUGA PRIYA, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting Held on 01.09.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of The Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of The validity
4. An annual status report should be submitted.
5. The TIREC will monitor The study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in The original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and The revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, The same should be documented.
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INTRODUCTION

Hypertensive disorders in pregnancy affects approximately 15% to 20% of all woman worldwide. The incidence is higher in western population and in woman belonging to high socio economic class. The incidence of gestational hypertension is on rise, which is highly attributed to life style changes, increased work stress, junk foods, and so on. Hypertensive disorders in pregnancy may be a sequelae of chronic hypertension (which exists even prior to pregnancy) or the ones which develop for the first time during pregnancy especially in the late second trimester. The severity of gestational hypertension varies from mild to severe gestational hypertension and finally eclampsia. This can affect both the mother and the fetus.

So there is a need to frame a good and effective screening test which can be used during routine antenatal check up to predict pre-eclampsia at the earliest. In this study, I have compared the lipid profiles of pre-eclamptic patients with normotensive patients. Various studies have been conducted previously on lipid profile changes in pre eclamptic female and have had a positive association between the same.

RESEARCH DEFINITION:

PRE ECLAMPSISA is New onset of hypertension with BP> 140/90 mmhg after 20 weeks of pregnancy with

a) .Protineuria- >300mg/24hrs (or) urine dipstick protein 1+. (OR)

b) Thrombocytopenia- platelet count<1,00,000

Renal insufficiency- Creatinine >1.1mg/dl

Liver involvement- serum transaminases elevated

Pulmonary symptoms

Cerebral symptoms

ABNORMALITY	NONSEVERE	SEVERE PE
DIASTOLIC BP	<110mmHg	>110 mmHg
SYSTOLIC BP	<160mmHg	>160 mmHg
PROTEINURIA	+	+
IMMINENT SYMPTOMS	-	+
SERUM CREATININE	Normal	Elevated
THROMBOCYTOPENIA	Absent	Present
FGR	Absent	Present
PULMONARY EDEMA	Absent	Present

CAUSES OF PRE ECLAMPSIA:

1. Placental implantation with abnormal trophoblastic invasion of uterine vessels.
2. immunological maladaptative tolerance between maternal, fetal, placental tissues.
3. Maternal maladaptation to cardiovascular & inflammatory changes of pregnancy.
4. Genetic factors including predisposing genes and epigenetic influences.

RISK FACTORS:

1. **RACE & ETHNICITY:**

Incidence of pre-eclampsia is 5% in whites, 9% in Hispanics, 11% in African American women.

2. **AGE:**

Young primi <20yrs, all patients > 35years have increased chances of pre eclampsia.

3. **PARITY:**

Primigravida have 15% more chances of pre eclampsia than multigravida.

4. **FAMILY HISTORY:**

Inheritance is three times increased if there is a affected first degree relative.

5. **PREVIOUS HISTORY OF PRE ECLAMPSIA:**

Women with pre elampsia in first have greater chances of recurrences in subsequent pregnancy.

6. WEIGHT:

Relationship between maternal weight & preeclampsia is progressive. It increases from 4.35% in women with BMI<20kg/m2 to 13.3% in women with BMI>35kg/m2.

7. MULTIPLE PREGNANCY:

Women with multiple pregnancy have 13% increased incidence of pre-eclampsia compared to 5% in singleton pregnancy.

8. DIET:

Deficiency in dietary calcium & vitamin C have increased incidence of pre eclampsia.

9. HYDATIDIFORM MOLE:

In large rapidly growing mole there is increased chances of pre-eclampsia.

10. RENAL DISEASE:

20% OF pre eclampsia occurs in pre existing renal disease patients.

11. THROMBOPHILIA:

Utero placental thrombosis is frequently associated with pre-eclampsia.

PATHOGENESIS:

Pre-eclampsia occurs only in pregnancy but it also occurs in pregnancy lacking fetus like molar pregnancy and in abdominal pregnancies suggesting that it is the presence of trophoblastic tissue that provides the stimulus for the disease. It's a two stage disease process which originates early in pregnancy:

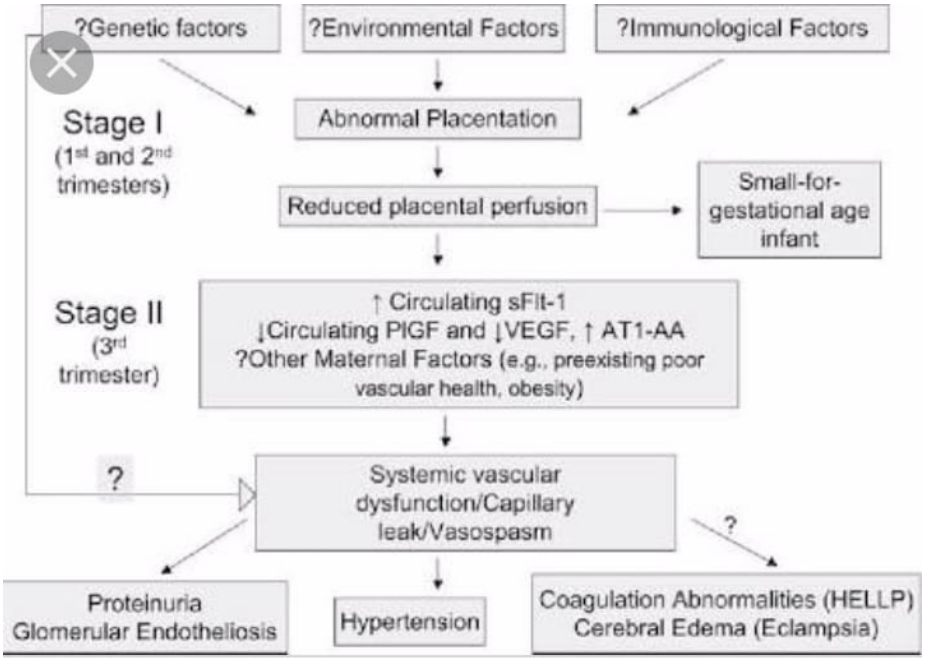
FIRST STAGE:

Trophoblast invasion is patchy and spiral arteries retain their muscular walls. It prevents development of high flow, low impedance circulation and leads to uteroplacentl ischemia.

SECOND STAGE:

Uteroplacental ischemia results in oxidative and inflammatory stress with involvement of secondary mediators of inflammation leading

to endothelial dysfunction, vasospasm and activation of coagulation system.



IMMUNOLOGIAL MECHANISM:

Genetic predisposition

↓

Abnormal trophoblast invasion

↓

Hypoperfused placenta

↓

Circulating factors

↓

Vascular and endothelial cell activation

There is increasing evidence both from animal and human studies that there is imbalance between angiogenic and anti angiogenic factors in pre eclampsia.

PATHOPHYSIOLOGY:

Pre eclampsia is a multi organ involvement with characteristic features in each organ system. The changes are either due to high blood pressure or diue to its effects like vasospasm, ischemia, endothelial dysfunction.

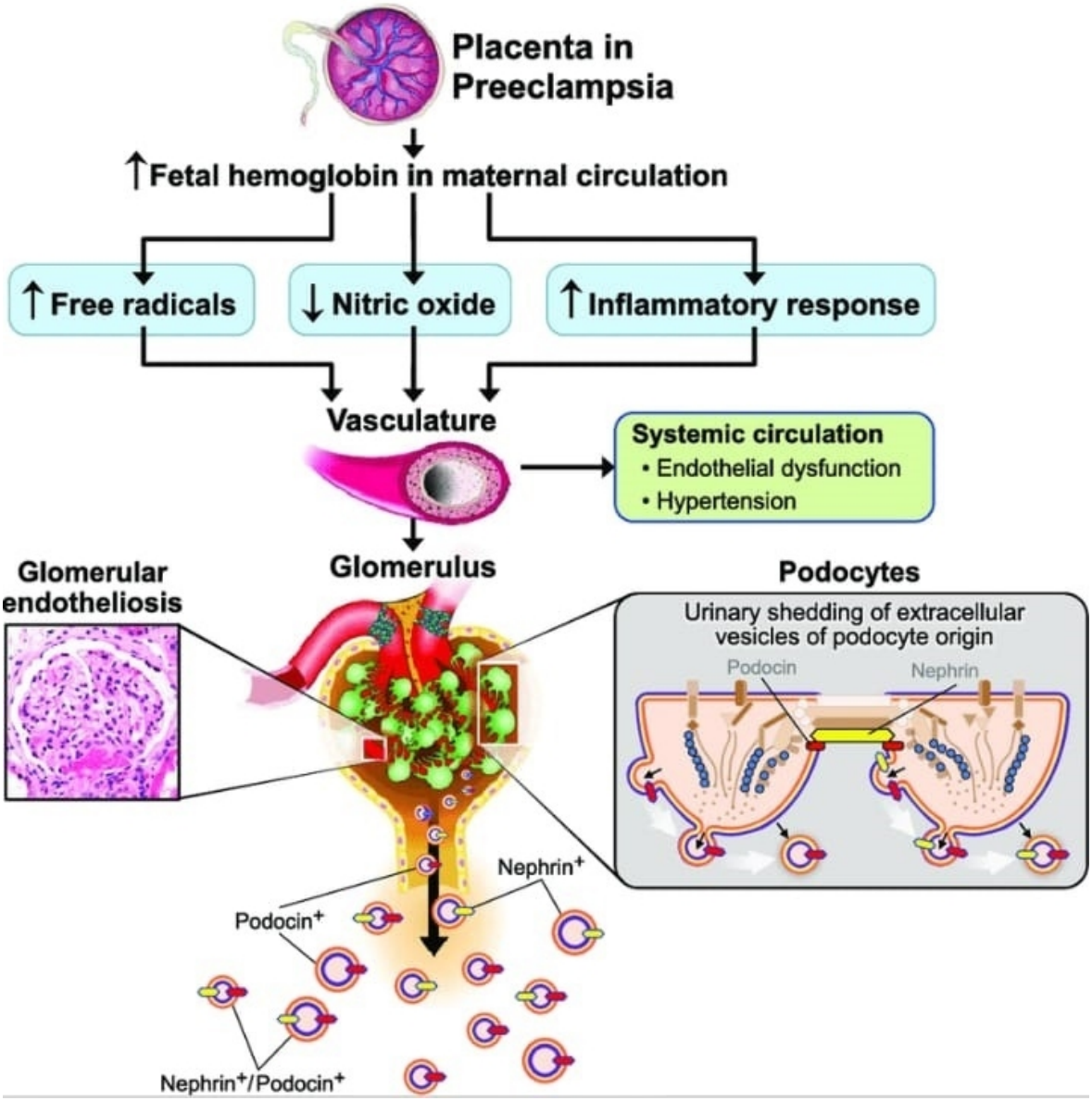
CARDIOVASCULAR SYSTEM:

The manifestation of pre-eclampsia are

a) Increased cardiac afterload
b) Endothelial activation with inter endothelial extravasation of intravascular fluid into extra vascular space.
c) Hyperdynamic ventricular function.
d) Increased peripheral resistance
e) Pulmonary edema due to alveolar endothelial – epithelial leak that is compounded by low oncotic pressure.

RENAL SYSTEM:

Following changes are noted as a consequence of Preeclampsia in kidney which are reversible



a) Glomerular capillary endotheliosis- glomeruli are enlarged and distended. Electron microscopy reveals endothelial cell

hyperplasia, exudation of macrophages, leucocytes into mesangium.

b) Focal segmental glomerulosclerosis
c) Nephrotic range of proteinuria
d) Acute tubular necrosis- usually reversible, occurs due to hypotension and hypovolemia in severe disease in association with obstetric hemorrhage.
e) Elevated GFR
f) Increased urinary sodium
g) Decreased urinary calcium due to increased reabsorption

LIVER:

Pathophysiological changes include ischemia, infarction and hemorrhage. It results in upper epigastric pain or right quadrant pain and tenderness usually in severe disease. Microscopic changes related to pre eclampsia in liver are 1. Periportal hemorrhage

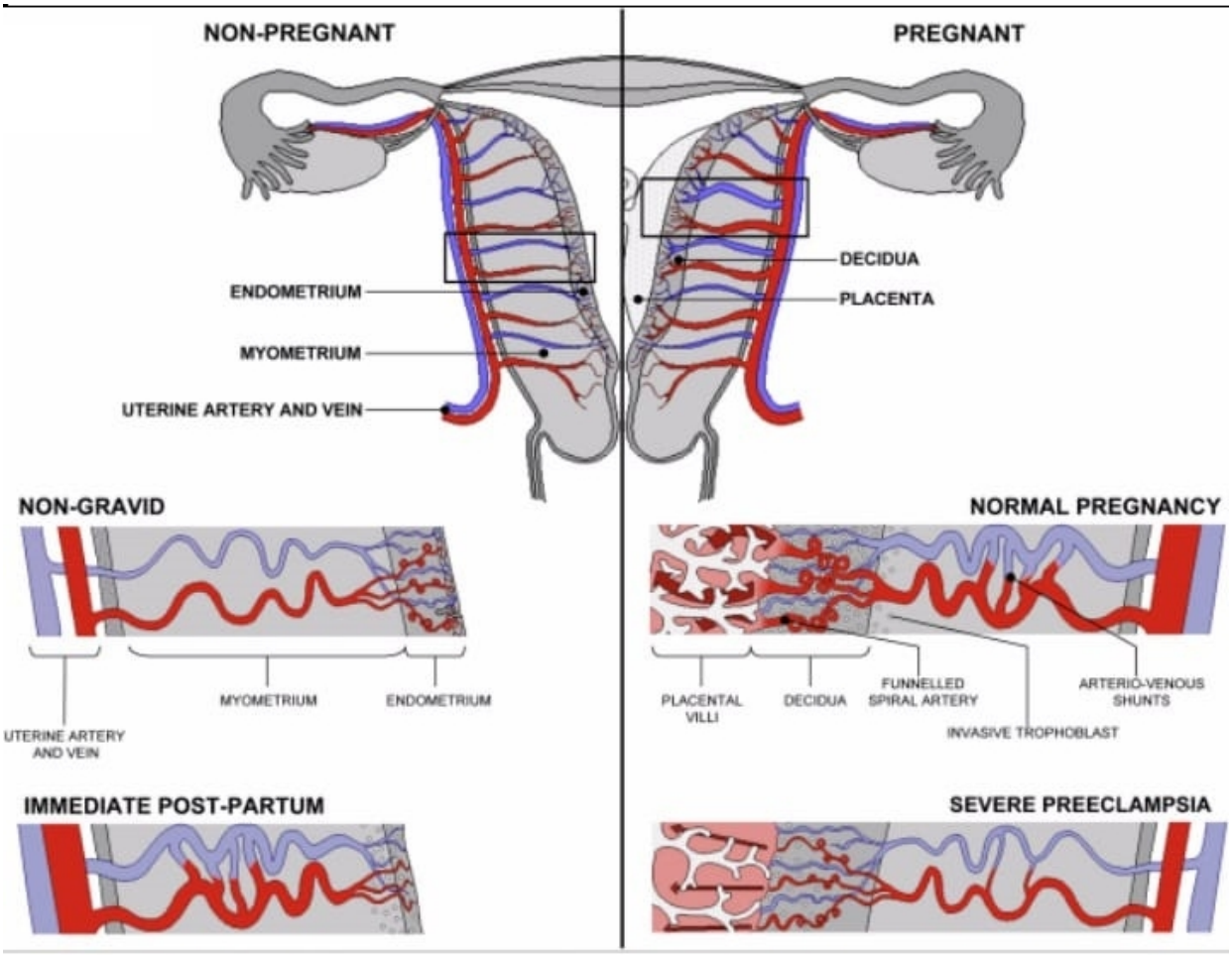
2. Hepatic hematoma

BRAIN:

Vasospasm and vasodilatationare both seen in pre eclampsia in cerebral arterioles. Due to severe hypertension there is vasoconstriction leading on to ischemia, cytotoxic edema and infarction. Once the regulatory mechanism fails there comes hyperperfusion and vasogenic edema. Common changes seen in brain are

a) Neurogenic edema
b) PRES
c) Blindness
d) Cerebral edema
e) Cerebral hemorrhage
f) Supra tentorial herniation
g) Foci of infarction

UTEROPLACENTAL CIRCULATION:



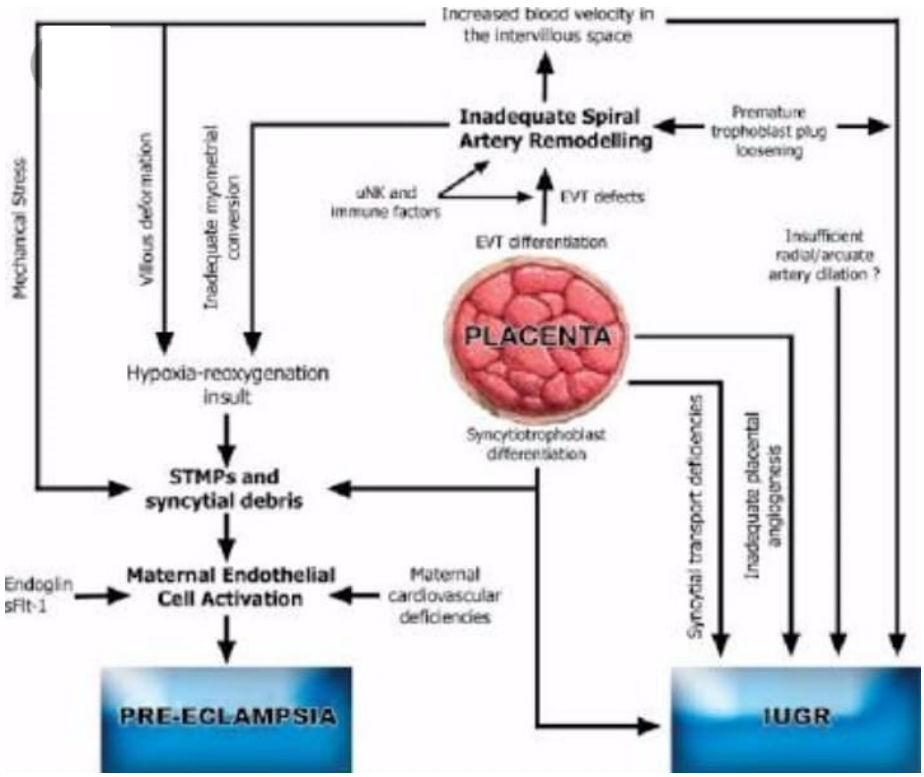
Compromised uteroplacental perfusion leads to increased perinatal mortality. The following Doppler abnormalities are seen during evaluation of pregnant mothers

a) Notching in uterine artery

b) Decreased placental vascularity index- Normal-15%, in pre-eclampsia it is 11.1%.

c) Increased resistance in spiral arteries.

PLACENTA:



There is increased incidence of infarcts, hematoma, congested chorionic villi, proliferative endarteritis and degeneration in placenta compared to normal placenta,. These changes are proportional to degree of severity. Microscopic examination reveals

a) Increased syncytial knots

b) Fibrinoid necrosis

c) Endothelial proliferation

d) Calcified and hyaline villous spaces.

COAGULATION SYSTEM:

Intravascular coagulation and erythrocyte destruction are consistent with pre-eclampsia. Increased factor 7 consumption and decreased anti thrombin 3, protein C &S are noted. Increased fibrinogen levels if associated with abruption. Fibronectin a glycoprotein linked to basement membrane is also found to be elevated in pre-eclampsia.

Findings pertaining to coagulation system in pre-eclampsia:

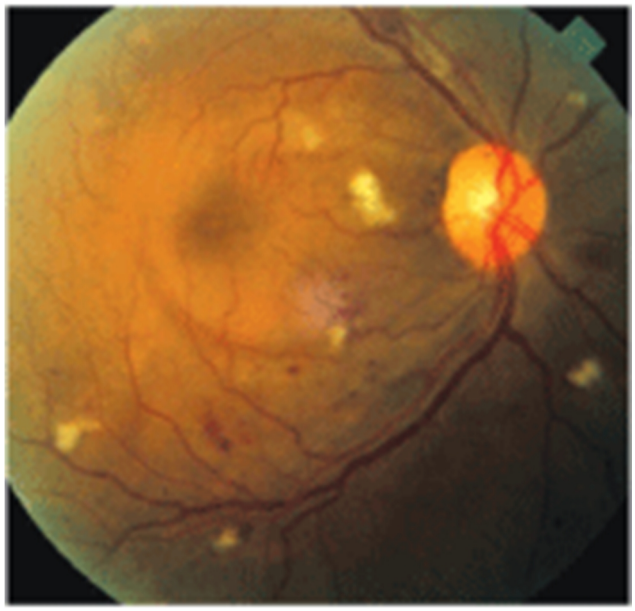
a) Thrombocytopenia

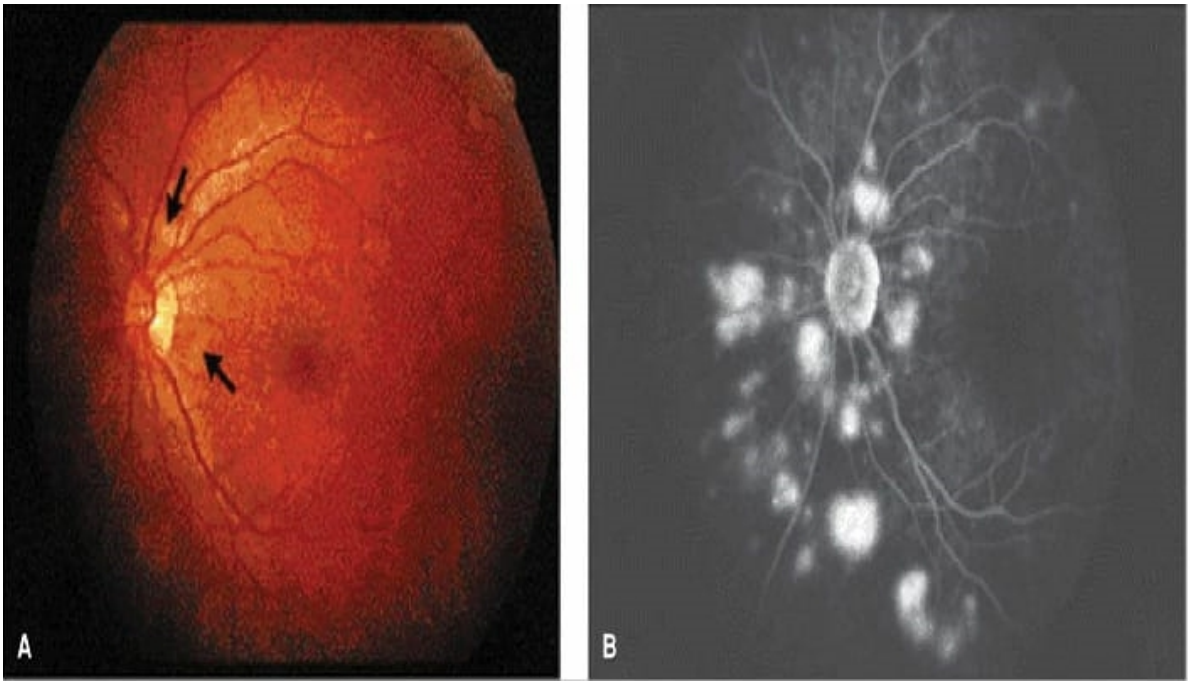
b) Elevated LDH

c) Increased coagulation factors

EYE:

Scotoma, blindness, diplopia commonly occurs during severe pre eclampsia. These symptoms usually resolves with anti hypertensive drugs and magnesium sulphate. Blindness usually arise from infarction of three susceptible areas in brain like occipital lobe, lateral geniculate nuclei and brain.





Occipital blindness usually occurs due to vasogenic edema. Rarely cerebral infarctions result in partial or total visual defect. Blindness from

retinal lesions usually occurs due to serous retinal detachment or retinal infarction called by a special name PURTSCHER RETINOPATHY.

PREDICTIVE TESTS FOR PRE ECLAMPSIA:

1) **PLACENTAL PERFUSION/ VASCULAR RESISTANCE-**

Roll over test - measures hypertensive response in women who are resting in decubitus position and then roll over to supine position. Increased blood pressure signifies positive test.

Cold pressor test – is a cardiovascular test performed by immersing hand into cold water for one minute and measuring changes in blood pressure and heart rate. Sensory afferent nerve triggers sympathetic activation leading to vasoconstriction. This results in elevated pulse pressure due to catecholamine release.

Isometric handgrip test - it activates mechanoreceptors due to increased muscle tension. It increases the excitatory state of the central nervous system and results in increase in sympathetic outflow and decrease in parasympathetic outflow thereby increase in blood pressure response.

Mid trimester Mean arterial pressure – Normal is 65-110mmHg. It is used similar to systolic blood pressure to monitor and treat

patients. Patients with MAP > 90mmHg is associated with higher risk of pre-elampsia

Uterine artery Doppler – Doppler interrogation of uterine artery shows end diastolic velocity and end diastolic notch. Notching is defined as a fall of atleast 50cm/sec from maximum diastolic velocity. It should be performed between 22 to 24 weeks of gestation. Notching demonstrates low positive predictive value for pre-eclampsia but 97% negative predictive value in high risk population. Predictive testing model including maternal factors,uterine artery pulsatality index in first trimester and change in pulsatality index between first and second trimester have a detection rate of 96%.

24 hours ambulatory BP measurement – helps to diagnose gestational hypertension at the earliest. It is a more sensitive predictor of cardiovascular outcome than conventional BP measurement. During daytime, more than fourteen measurements and at night time seven measurements are made.

2) RENAL DYSFUNCTION:

Uric acid – Increased serum uric acid in pre-eclampsia is likely to be influenced by changes in maternal renal function like decreased tubular reabsorption and increased production. Also placenta and fetus play a role in uric acid elevation. Hypoxia induced change in production of xanthine oxidase/ dehydrogenase.so uric acid may be a better marker of poor placental perfusion and fetal hypoxia.

Microalbuminuria - Urinary protein values of more than 300mg/24hrs or urinary dipstick values of more than 1+ suggest significiant protein excretion in pregnancy. Acute onset of proteinuria and worsening of proteinuria is a marker of poor maternal outcome in pregnant hypertensive mothers.

Urinary calcium/kallikrein

Microtransferrineuria

Fasting urine albumin/creatinine ratio

3) FETAL PLACENTAL UNIT:

Human chorionic gonadotrophin

Alpha feto protein

Pregnancy associated placental protein A

Placental protein 13

Inhibin

Marker	Mechanism of action	Levels associated with PE
PAPPA-A	Insulin like growth factor binding protein protease: impaired trophoblast invasion and fetal cell growth	↓
PIGF	Vascular endothelial growth factor trophoblastic implantation	↓
sFlt-1	Antiangiogenic factor	↑
Inhibin A & B	Maintenance of spiral artery	↓
sEng	Impairs binding of transforming growth-	↑

	β1 to cell surface receptors, inhibiting angiogenesis	
PP13	Binds to protein on extracellular matrix between placenta and myometrium: placenta implantation & remodeling	↓
ADAM12	Placenta derived multidomain glycoprotein: fetal & placental growth	↓

4) ENDOTHELIAL DYSFUNCTION/ OXIDATIVE STRESS:

Platelet count

Antiphospholipid antibody

Lipid profile

Fms like tyrosine kinase receptor-1

Homocysteine

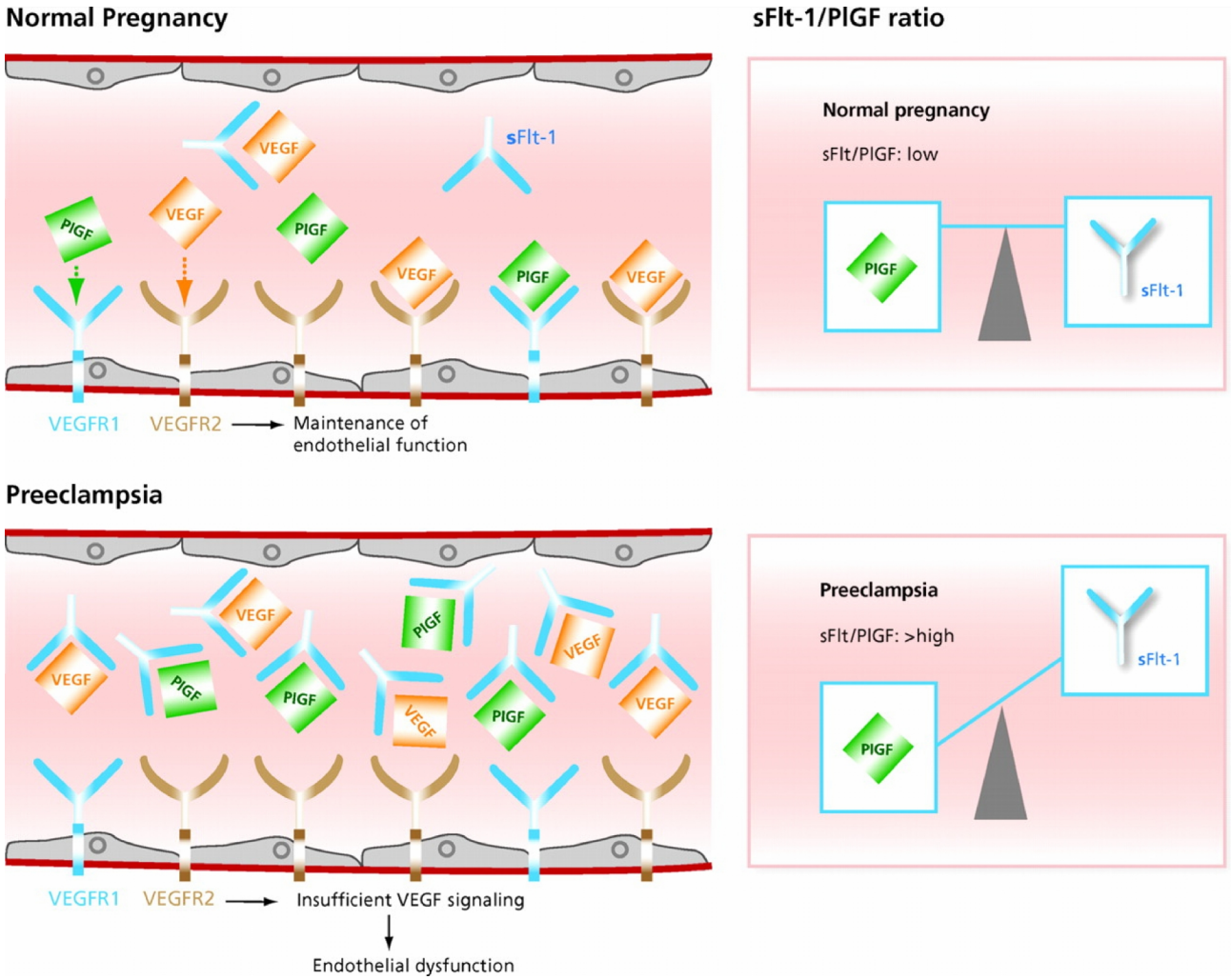
Beta2 microglobulin

Fibronectin

VEGF, PIGF.

Endoglin

Cell adhesion molecules



5) **Genetic markers**

The candidate genes selected to predict pre eclampsia are

a) **MTHR- Methylene tetra hydrofolate reductase**

b) **F5- factor 5 leiden**

c) **AGT- angiotensinogen**

d) **ACE- Angiotensin converting enzyme**

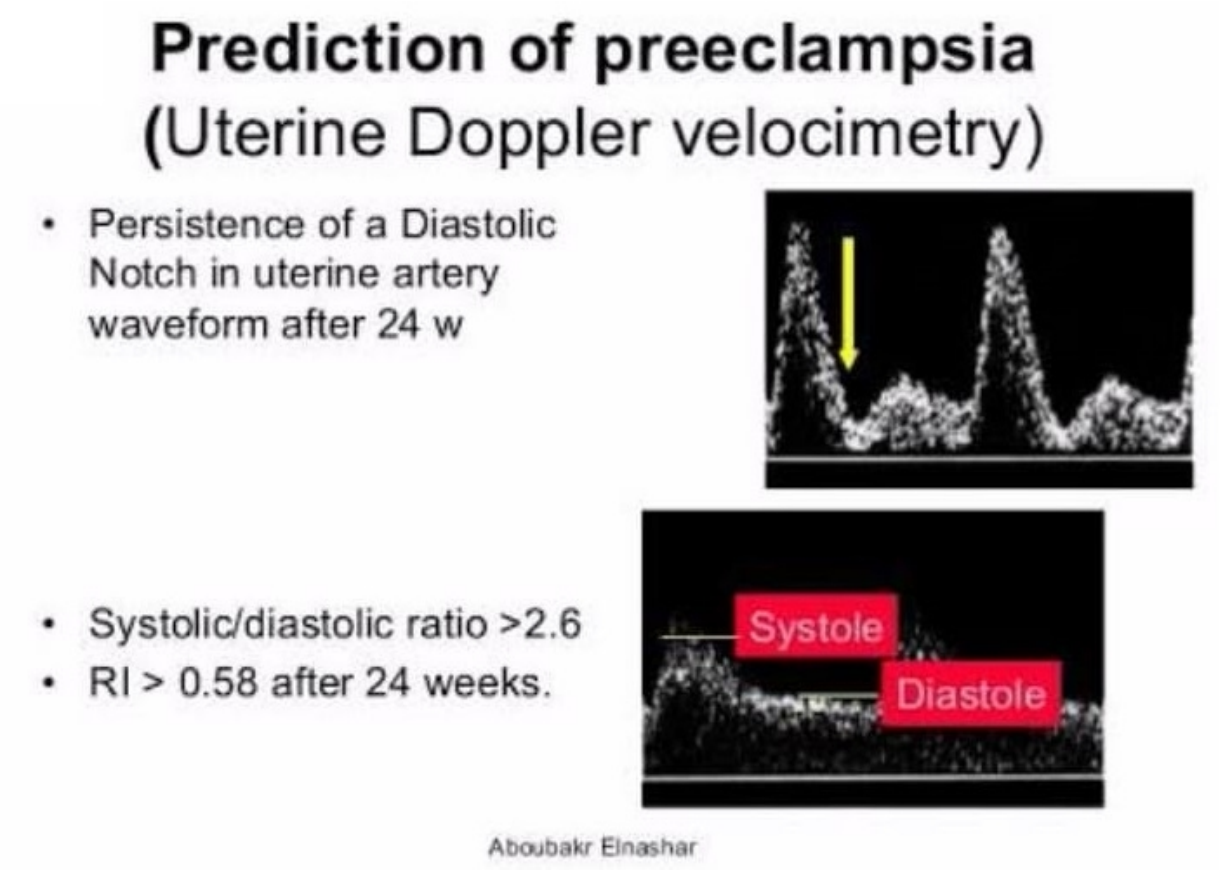
e) **SERPINE 1- Serine peptidase inhibitor**

f) **LPL- lipoprotein lipase**

g) **NOS 3- nitric oxide synthetase**

h) **F2- Prothrombine**

6) **Cell free DNA**- Due to increased maternal and fetal trafficking in pre-eclampsia there is more number of fetal cells detected in maternal blood which can be studied to predict hypertensive disorders earlier in pregnancy.



Though a lot of tests have been studied so far, no tests is found to better and cost effective in predicting pre-eclampsia. BMI, Alpha fetoprotein, fibronectin, uterine artery Doppler seemed to have a good specificity of 90% but poor sensitivity. Many tests should come to predict pre eclampsia in early pregnancy sonthat there will be a better perinatal and maternal outcome.

METHODS TO PREVENT PRE ECLAMPSIA:

Few methods used to prevent or modify the severity of pre-eclampsia are

1) Dietary modification :

    Salt restriction

    Calcium supplementation

    Fish oil supplementation

    Arginine supplementation

    Increased protein intake

    Change in dietary habits

2) Exercise :

    Physical activity

    Stretching

    Aerobic exercise

3) Cardiovascular drugs :

    Diuretics

    Anti hypertensive drugs

4) Anti thrombotic drugs

Low dose aspirin used for a better perinatal outcome.50-150mg/day is advised as it selectively inhibits TX-A2 synthesis with minimal effect on prostacycline. CLASP (Collabrative low dose aspirin) study is the largest conducted so far to prove the efficacy of aspirin in reducing the morbid effects of pre elampsia.

5) Magnesium, Folic acid, Vitamin B Supplementation

6)Heparin in women with anti phospholipid syndrome.

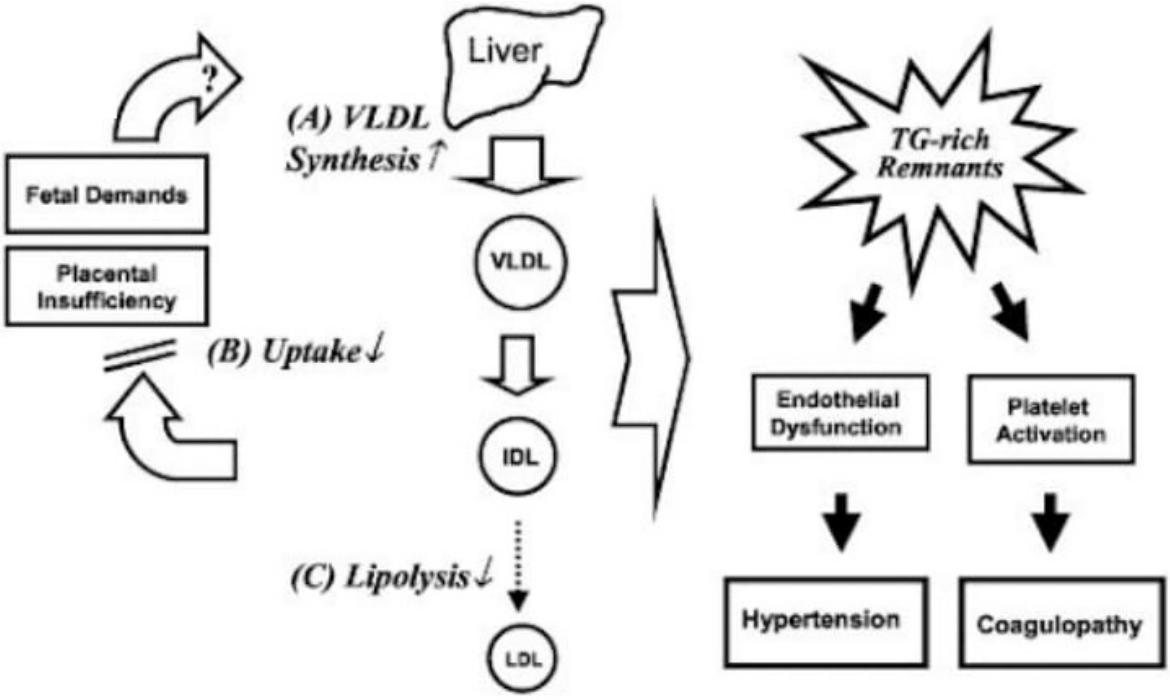
7)ANTI OXIDANTS: Vitamin C, E, lycophene,N acetyl cysteine, garlic supplementation in preventing pre eclampsia as studied by a Cochrane review in 2008.

7) Bed rest.

8) Control of body weight

LIPID METABOLISM IN PREGNANCY:

Abnormal lipid profile is known to be strongly associated with atherosclerotic cardiovascular disease and has a direct effect on endothelial cell activation. Abnormal lipid metabolism seems to be important in the pathogenesis of preeclampsia too.



Normal human pregnancy results in a pronounced physiologic hypertriglyceridemia involving a gestational rise in blood triglycerides and cholesterol. Serum triglycerides and LDL concentrations in women with preeclampsia were higher than those in women with uncomplicated pregnancy.

During the first trimester there is increased maternal fat accumulation (anabolic state) is presumed to be important for the subsequent hypertriglyceridemia normally occurring in later gestation (catabolic state). Circulatory concentrations of VLDL & LDL normally increase with gestational age as reflected by marked increases in serum triglycerides and cholesterol. The hypertriglyceridemia is due primarily to enhanced entry of triglyceride rich lipoproteins (especially VLDL) into the circulation rather than to diminished removal. Estrogen may play a major role in the lipoprotein patterns seen in human pregnancy although LDL cholesterol is more influenced by the combined effect of increased estrogen and progesterone. Additionally placental lipoprotein lipase activity normally increases as term approaches.

The mechanisms driving the abnormal elevation in triglycerides and free fatty acids in preeclampsia are unclear metabolic patterns resembling `Syndrome X' or `Insulin Resistance Syndrome'. Heightened insulin resistance occurring in preeclampsia would increase fatty acid mobilisation from the visceral fat, promote overproduction of VLDL by the liver and suppress activity of posthepatic lipoprotein lipase resulting in elevated serum free fatty acids and triglycerides.

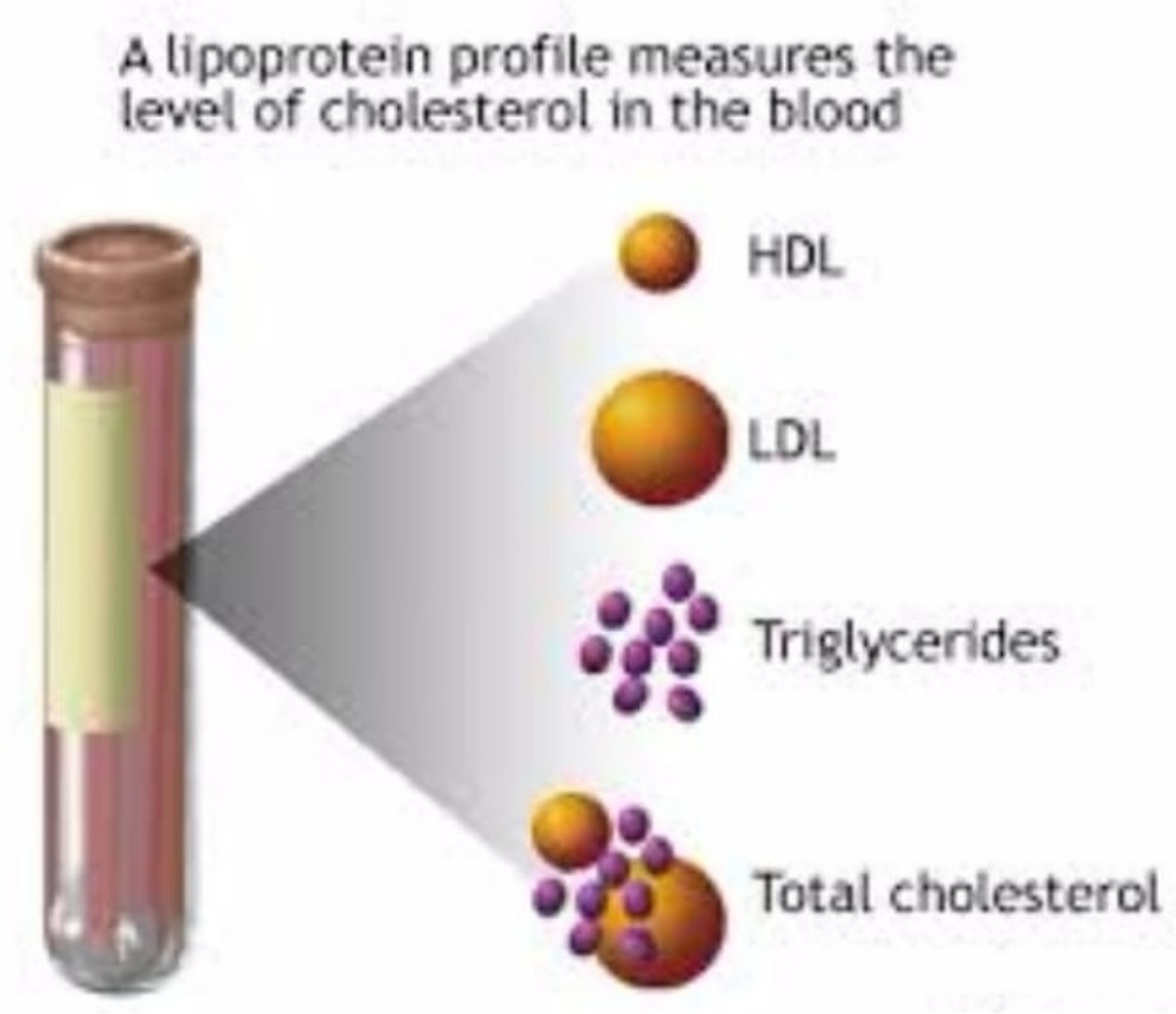
MECHANISM BEHIND LIPID METABOLISM:

1. Enhanced lipolysis in adipose tissue facilitating availability of substrates for Triglyceride synthesis and VLDL influx into circulation.
2. Increased chylomicron formation from dietary lipids.
3. Reduced lipoprotein lipase activity in extrahapatic tissues resulting in decreased triglyeride removal

COMPONENTS OF LIPID PROFILE:

Lipid profile or lipid panel includes the following components:

1. Total cholesterol
2. Triglycerides
3. LDL
4. HDL
5. VLDL



Testing lipid profiles require about 9-10 hours of fasting initially. But now most of the labs use non fasting state to test it without affecting the efficacy of results.

Fasting blood samples are analysed for total cholesterol, triglyceride, HDL by enzymatic methods using semi auto analyser. Serum LDL cholesterol value is calculated by FREDERICKSON – FRIEDWALD formula .According to this,

LDL = Total cholesterol – (HDL + VLDL)

Serum VLDL is calculated by dividing triglyceride by five

Using these values ,we can make TC/HDL, TGL/HDL, HDL/LDL ratios which can be used to assess dyslipidemia,.

REVIEW OF LITERATURE

Carl Hubel et al., (1995) in their study, concluded that triglycerides and fatty acids, but not cholesterol, are increased in preeclampsia and correlate with the lipid peroxidation metabolite malondialdehyde 5. These interactions contribute to endothelial cell dysfunction in preeclamptic patients.

Ray et al., (2006) studied the risk of preeclampsia in the presence of maternal triglyceridemia, another major element of the metabolic syndrome.24 A total of 19 case control and 3 prospective cohort studies were included. In 14 studies, the mean TGL concentration was significantly higher among preeclamptic cases than among unaffected controls. In seven other studies, there was a nonsignificant trend in the same direction. The risk of preeclampsia typically doubled with each increasing TGL category. In the four studies, that adjusted for potential confounders, such as maternal age, parity and body mass index, there was a four fold higher risk of preeclampsia in the highest relative to the lowest triglyceride category.

Thus, the study concluded that there exists a consistent positive association between elevated maternal triglycerides and the risk of preeclampsia. Given that maternal hypertriglyceridemia is a common

feature of the metabolic syndrome, interventional studies are needed to determine whether pre-pregnancy weight reduction and dietary modification can lower the risk of preeclampsia.

Sattar et al., (2002) studied the involvement of lipids in the pathophysiology of preeclampsia by doing lipid profile and lipoprotein subfactors in preeclampsia and in normal pregnancy.25 They showed the potential links of the atherogenic lipid profile in preeclampsia to oxidative pathways, endothelial dysfunction and vascular inflammation.

Manten et al., (2004) studied the role of lipids in the endothelial cell dysfunction and thus leading to preeclampsia.18 Seventy women were recruited, 50 were nulliparous women with singleton pregnancies and not in labour, 10 had severe preeclampsia. 20 had mild preeclampsia and 20 were healthy pregnant controls. The other 20 women were healthy non pregnant controls. There was a tendency to higher Lipoprotein-a concentration in women with preeclampsia. In the preeclampsia group, the concentrations of cholesterol and triglycerides were reported to be higher and the HDL cholesterol concentration was lower as compared to normal pregnancy.

Gratacos et al., (2003) evaluated the susceptibility to oxidation of LDL in women with a history of preeclampsia by a case control study.9

Thirty five patients, who were diagnosed with severe preeclampsia and 35 controls were matched for age, BMI, smoking and parity. Plasma samples were analysed for total cholesterol, HDL, LDL, triglycerides and lipoprotein A. The in vitro susceptibility to oxidation of LDL was measured and expressed in minutes.

Mean LDL cholesterol and triglyceride levels were higher in preeclampsia group compared with controls. The susceptibility to oxidation of LDL was also significantly higher in the preeclampsia group.

Kaaja et al., (1995) and Sattar et al., (1997) have found higher plasma TGL and low HDL concentrations in women with preeclampsia and gestational HT.12,26 Given these findings, it had been postulated that lipid abnormalities could play a role in the pathogenesis of preeclampsia, causing altered endothelial cell dysfunction and vascular damage.

Sattar et al., (1997) studied the lipoprotein subfraction concentrations in preeclampsia.26 They concluded that the pathogenesis parallels to atherosclerosis.

In a recent study, in 2001, The National Heart Foundation of Australia found a correlation between F2 - isoprotanes with increased levels of total and LDL cholesterol and triglycerides and significantly

reduced levels of HDL cholesterol in women with preeclampsia, compared with that of the normotensive group. Increased levels of F2 isoprotanes show that preeclamptic subjects are under increased oxidative stress, indicated by the increased rates of neutrophil activation.

Anceschi et al., (1992) conducted a study on erythrocyte membrane composition in pregnancy induced hypertension, which is an evidence for an altered lipid profile.1

They concluded that the cholesterol / phospholipid ratio was significantly higher in the women with pregnancy induced hypertension compared with the normotensive pregnant women (PIH - 1.24 compared with normotensive - 0.88). The increased cholesterol / phospholipid ratio of the erythrocyte membrane found in pregnancy induced hypertension represents one factor involved in the pathophysiology of this condition and a possible biochemical marker of the disease.

In a study by Dempsey et al., (2004), maternal birth weight was correlated negatively with triglycerides and correlated positively with high density lipoprotein cholesterol.7 Women who weighed <2500 gms at birth had higher triglyceride and total cholesterol concentration and lower HDL concentrations, when compared with women who weighed 3000 to 3499 gms at birth. Women who were born small and

became overweight in adulthood had less favourable lipid profiles than their counterparts who weighed >2500 gms at birth and remained lean.

Thus they concluded that factors that are related to growth in utero may help to predict the subsequent risk of altered lipid metabolism during pregnancy which may, in turn be causally related to the occurrence of preeclampsia.

In a study by Barden et al., (1999) the aim was to identify those factors in the non pregnant state that distinguished women who developed preeclampsia from those who had normotensive pregnancies.2 The results were : Regardless of parity, women with preeclampsia had elevated BMI before, during and after pregnancy compared with women who had normotensive pregnancies. Triglycerides were significantly elevated in women who had preeclampsia both before and after delivery.

The relative elevation of blood pressure, BMI and lipids in the non pregnant state are features of the metabolic syndrome and may be important sensitising factors contributing to the pathogenesis of preeclampsia. A familial predisposition to preeclampsia may operate partly through these mechanisms.

Kokia et al., (1999) studied the maternal serum lipid profile in pregnancies complicated by hypertensive disorders.15 Serum

triglyceride levels were significantly elevated in the hypertensive patients. This elevation was not influenced by the severity or etiology of the hypertension. The lipid profile found in the hypertensive pregnant patients could be associated with enhancement of pathological lipid deposition in predisposed vessels such as the uterine spiral arteries. Further the hypertriglyceridemia found in the hypertensive patients may be associated with the hypercoagulability reported in pregnancy induced hypertension.

Daniel et al., (2004) investigated the relationship between early pregnancy plasma lipid concentration and risk of preeclampsia.6 They concluded that early pregnancy dyslipidemia is associated with an increased risk of preeclampsia. This association may be significant in understanding the pathologic processes of preeclampsia and may help in developing strategies for prevention or early diagnosis of the disorder.

Thadhani et al., (1999) and Bodnar et al., (2005) found an association between high body mass index and hypercholesterolemia, which in turn increases the risks of preeclampsia.28, 4

Wolf et al., (2001) showed that obesity has a role to play in preeclampsia.33

Ramsay et al., (2002) inferred that obesity is associated with dysregulation of metabolic, vascular and inflammatory pathways.23 O'Brien et al., (2003) found that increased maternal BMI increases the risk of preeclampsia.22

Torun et al., (2001) found that dyslipidemia in early second trimester is mainly a feature of women with early onset pre-eclampsia.29 Hubel et al., (1998) found elevated levels of low density lipoproteins and vascular cell adhesion molecule -1 in association with hyperlipidemia in preeclampsia.11

Ware Jauregui et al., (1999) compared the plasma lipid concentrations in preeclamptic and normotensive Peruvian women.32 Vanderjagt et al., (2004) found that high - density lipoprotein and homocysteine levels correlated inversely in preeclamptic women.30

Kharba et al., (1998) determined the lipid peroxidation and vitamin E levels in preeclampsia.14 Maseki et al., (1981) compared the lipid peroxide levels and lipid content of serum lipoprotein fractions of normotensive pregnant subjects and the subjects with preeclampsia.19

Gratacos et al., (1996) studied the variation in lipid levels during pregnancy in women with different types of hypertension.10 Llurba et al., (2004) and Wakatsuki et al., (2000) did a comprehensive study of

oxidative stress and the antioxidant status in preeclampsia and normal pregnancy.16,31 Granger et al., (2001) linked endothelial dysfunction with placental ischaemia, leading to the pathogenesis of preeclampsia.8

Sattar et al., (1997) studied the threshold effect of plasma triglyceride on appearance of small dense low density lipoproteins as well as the lipoprotein subfraction changes in normal pregnancy.27 Lorentzen et al., (1995) found the fatty acid pattern of esterified and free fatty acids in sera of women with normal and preeclamptic pregnancy.17

Mikhail et al., (1995) showed the relationship between plasma triglyceride levels and severity of preeclampsia.20 Khaliq et al., (2004) studied the serum lipid and lipoproteins in preeclampsia with special reference to parity.13 Murai et al., (1997) correlated the maternal and fetal modulators of lipid metabolism with the development of preeclampsia.21

AIM

To analyse the lipid profile in normal pregnant women and the lipid profile changes in women with pre eclamptic mother .

MATERIALS AND METHODS

Study Design

Comparative Study.

Study period

September 2017 to August 2018.

Objective:

1.To analyse lipid profile changes in pre eclamptic mother.

2.TO compare the lipid profile values of normal pregnant mother and pre eclamptic mother.

3.To establish the relationship between lipid profile changes and pre eclampsia.

4.To know the efficacy of lipid profile in predicting pre eclampsia.

Inclusion Criteria

1. Pregnant women in the third trimester diagnosed as Pre eclampsia with no other associated complications.
2. Normal women in the third trimester of pregnancy with no other maternal medical complications, admitted for safe confinement.

Exclusion Criteria

- Chronic hypertension
- Pregestational Diabetes mellitus
- Nephrotic Syndrome
- Cardiac Disease.
- Hepatic Disease.
- Twin pregnancy
- Any medications except for vitamins and minerals.
- Smoking
- Alcohol abuse
- Labour contractions
- Thyrotoxicosis.
- Molar pregnancy

Methodology

All pregnant mother included in the study are subjected to detailed history taking, height, weight, blood pressue measurement. Antenatal check up was done. 5ml of venous blood is drawn for analysis.

Fasting blood samples were taken from 100 pregnant patients with pre eclampsia and 100 normal pregnant women, admitted in Tirunelveli Medical College Hospital.

The samples were subjected to analysis of lipid profile.

The patients were also followed up till delivery to assess the outcome of both mother and the baby.

Specimen

Freshly collected plasma. Anticoagulant used was EDTA.

Procedure

The lipid profile of the samples were determined using a semiautomated analyser.

Reagents used :

The following reagents were used for determination of the lipid values.

Total Cholesterol: Cholesterol Oxidase, Peroxidase

HDL Cholesterol: Phosphotungstate / Magnesium precipitation

LDL Cholesterol: Catalase / cholesterol esterase / cholesterol oxidase

Triglycerides: Glycerol Phosphate oxidase/ Peroxidase

Reference values of the lab

Total cholesterol: 125 - 200 mgs / dl HDL

HDL : 30 - 65 mgs / dl

Triglycerides : 25 - 200 mgs / dl LDL

LDL : 85 - 130 mgs / dl

VLDL : 5 - 40 mgs / dl.

LIPID PROFILE

	DESIRABLE	**BORDERLINE**	**HIGH RISK**
Cholesterol	<200 mg/dl	200-239 mg/dl	240 mg/dl
Triglycerides	<150 mg/dl	150-199 mg/dl	200-499 mg/dl
HDL cholesterol	60 mg/dl	35-45 mg/dl	<35 mg/dl
LDL cholesterol	60-130 mg/dl	130-159 mg/dl	160-189 mg/dl
Cholesterol / HDL ratio	4.0	5.0	6.0

RESULTS OF THE STUDY

The women with pre eclampsia were grouped as Group I (Study Group).

The normal pregnant women were grouped as Group II (Control Group). The results of the study were analysed as follows

The results were analysed between group I and group II according to the age distribution, booked / unbooked status, obstetrics score, history of PIH in the previous pregnancy, body mass index, total cholesterol, HDL, VLDL, LDL, triglycerides, triglyceride levels.

Data will be analysed using student "T" TEST and significance will be expressed in p values.

TABLE - 1

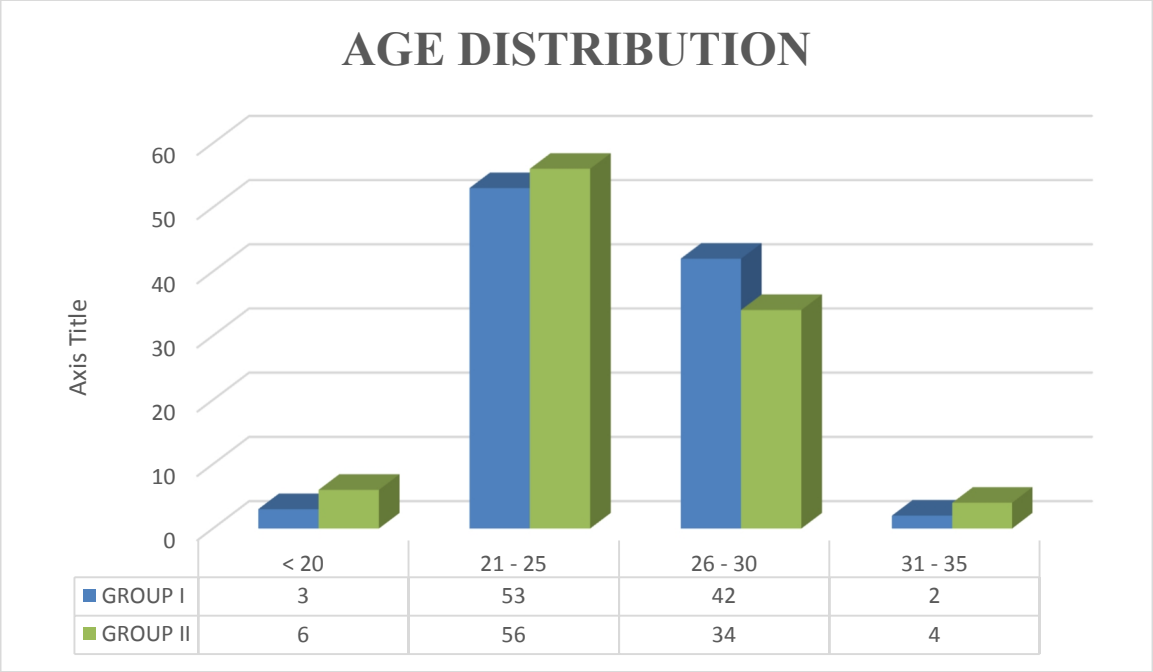
AGE

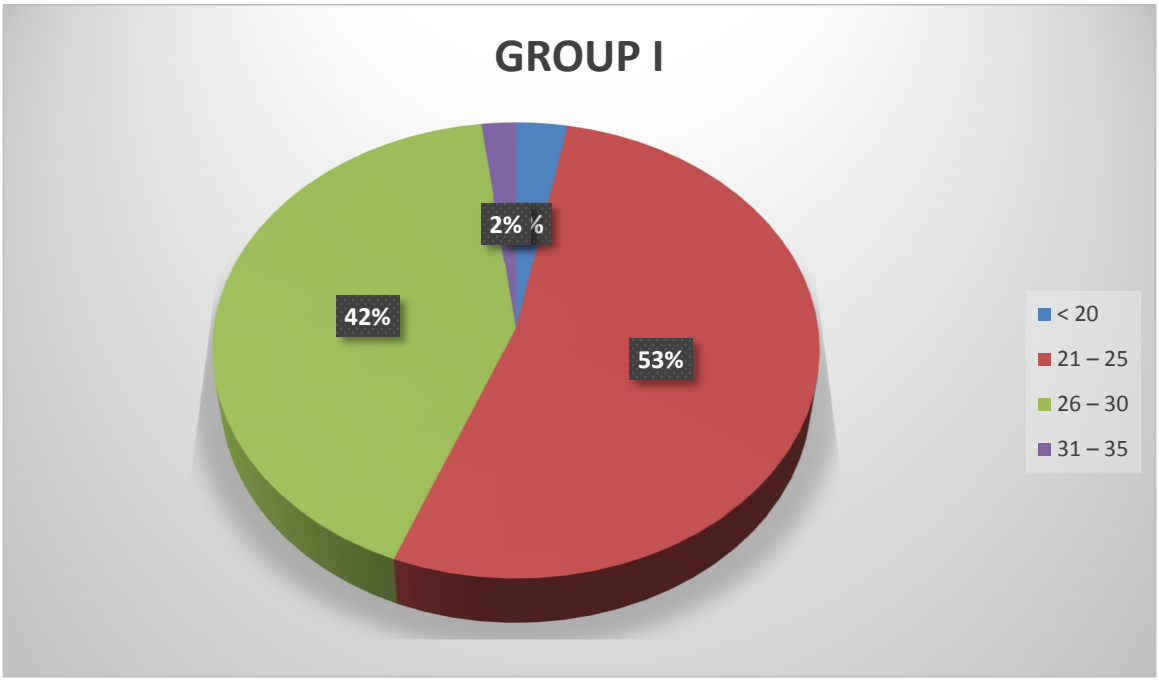
	No. OF	MEAN AGE		S.E. OF
GROUP I	100	25.1700	2.816	0.28817
GROUP II	100	24.6100	3.03147	0.30315

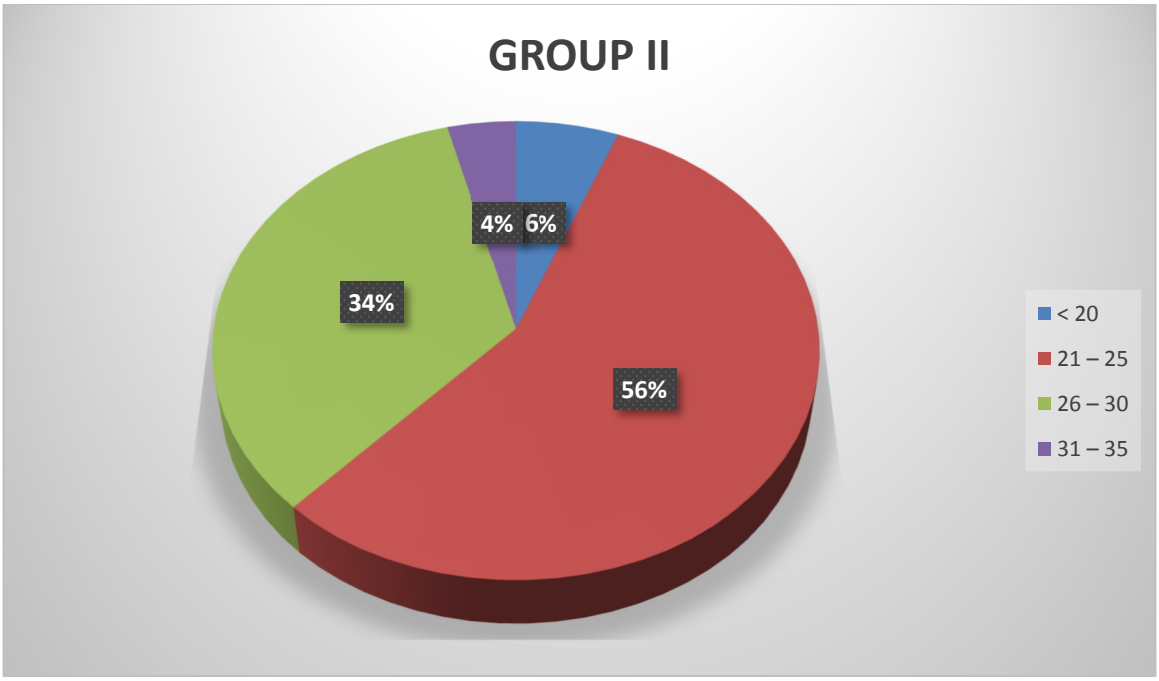
t = 1.330; p = 0.182 Not significant

TABLE - 1(a) AGE DISTRIBUTION

AGE IN YEARS	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
< 20	3	3	6	6
21 – 25	53	53	56	56
26 – 30	42	42	34	34
31 – 35	2	2	4	4
Total	**100**	**100**	**100**	**100**


AGE DISTRIBUTION
Axis Title
60
50
40
30
20
10
0
< 20
21 - 25
26 - 30
31 - 35
GROUP I
3
53
42
2
GROUP II
6
56
34
4


GROUP I
2%
42%
53%
< 20
21 – 25
26 – 30
31 – 35



Inference

There is no significant change in the age distribution between the two groups.

53% of the patients in Group I and 56% of patients in Group II were in the age group of 21 - 25 years.

Only 2% of patients in Group I and 4% of patients in Group II were in the age group of 31 - 35 years.

TABLE - 2

BOOKED / UNBOOKED

BOOKING STATUS	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
Booked	92	92	92	92
Unbooked	8	8	8	8
Total	**100**	**100**	**100**	**100**

p = 100 ; x2 = 0.000 Not significant

BOOKED / UNBOOKED

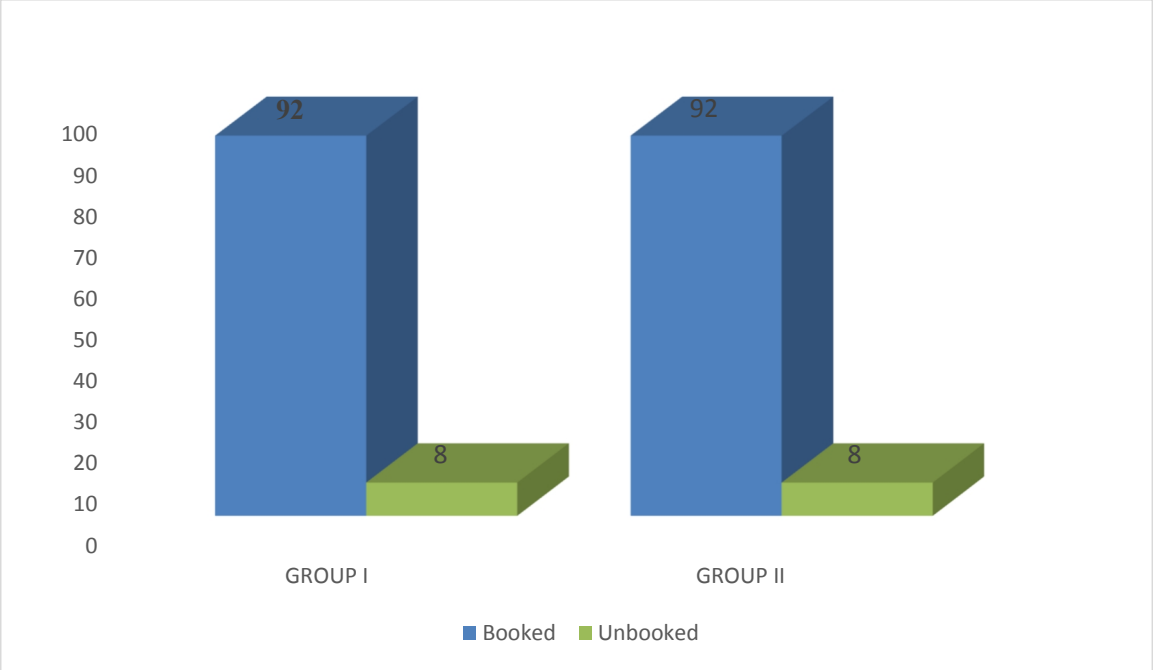
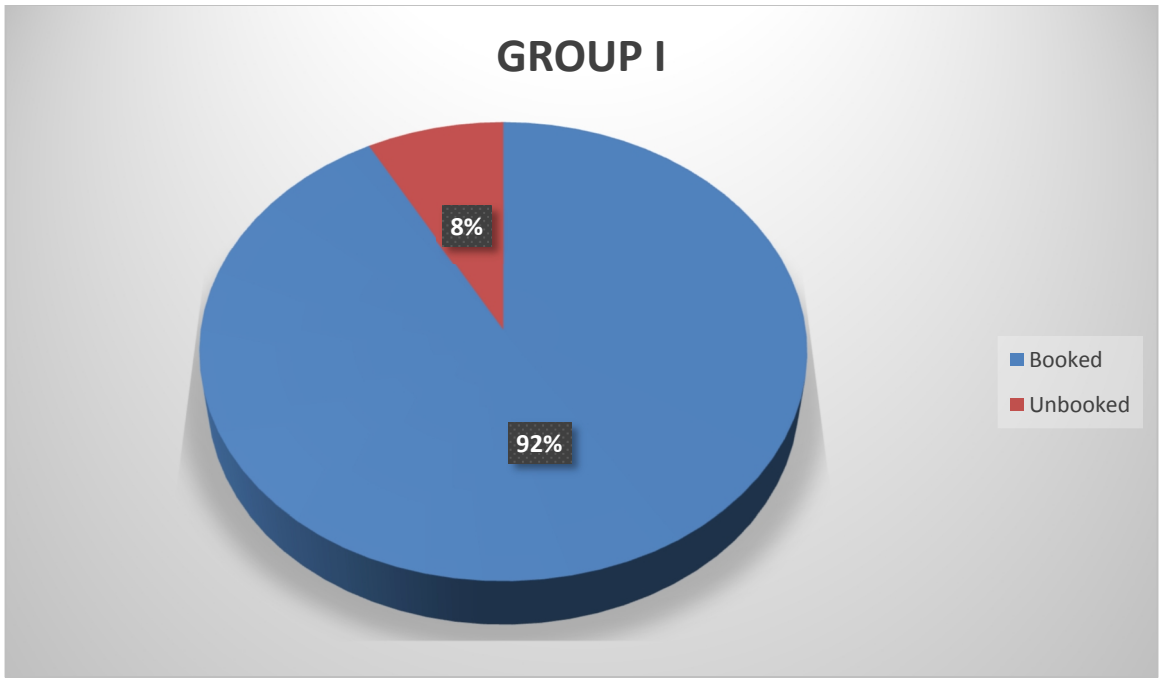
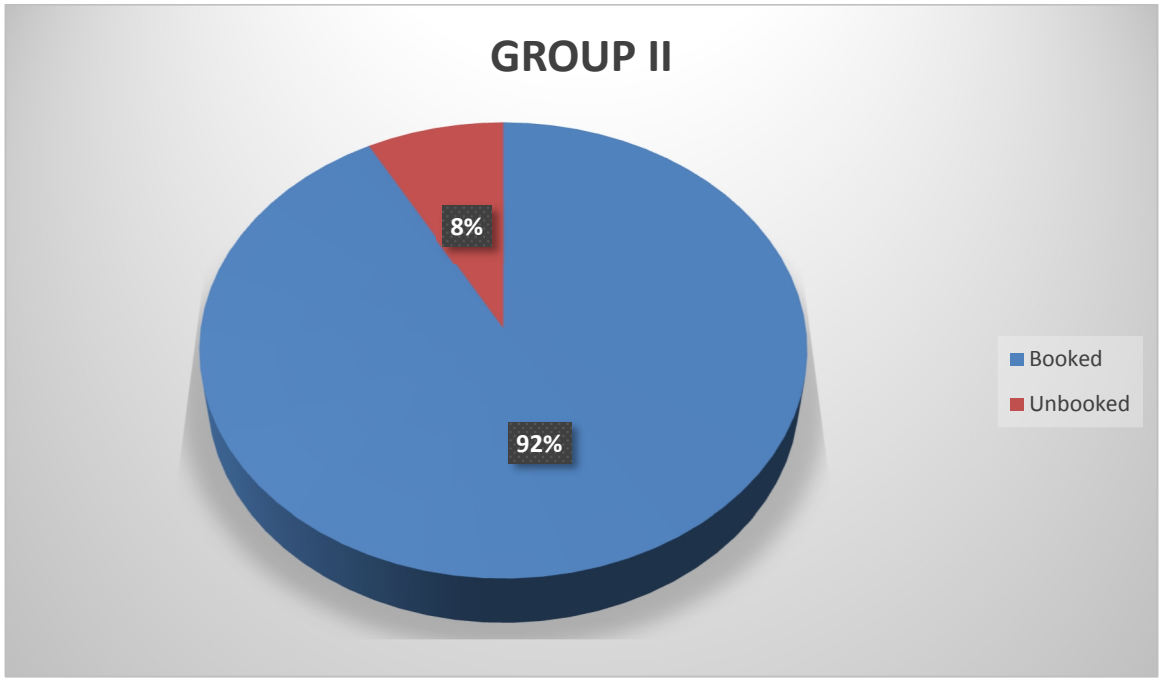


Fig. 2


GROUP I
8%
92%
Booked
Unbooked


GROUP II
8%
92%
Booked
Unbooked

Inference

The booking status of both the study group and the control group were the same, 92% being booked and 8% being unbooked.

TABLE - 3

OBSTETRIC SCORE

GRAVIDA	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
1	47	47	51	51
2	42	42	38	38
3	7	7	9	9
4	4	4	2	2
Total	**100**	**100**	**100**	**100**

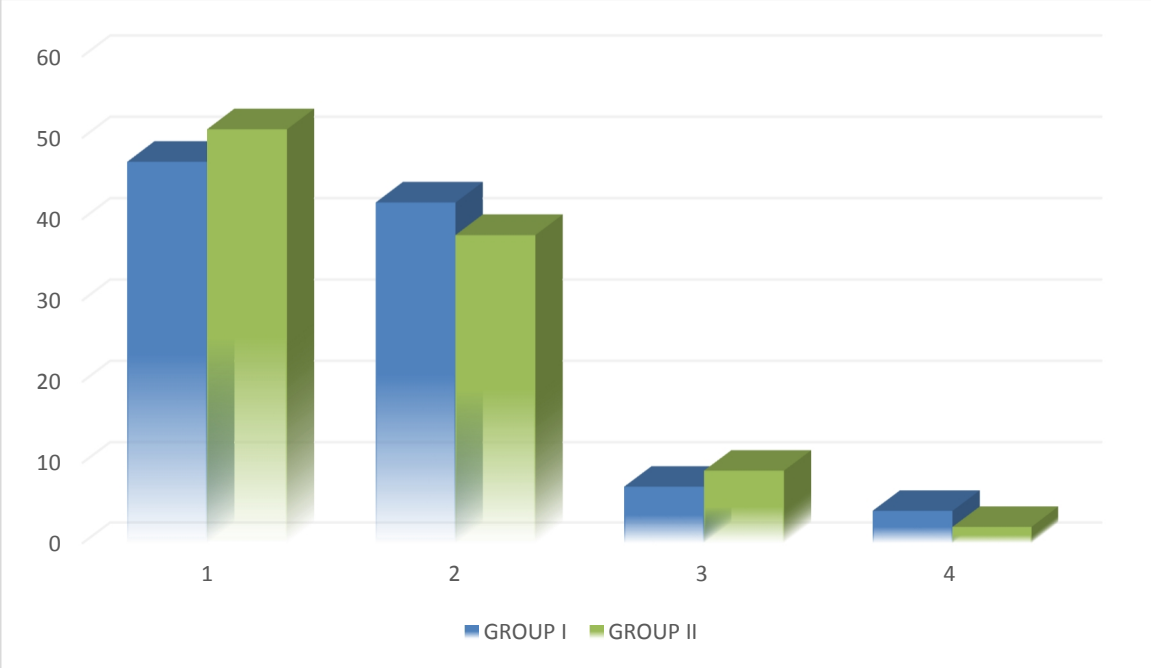
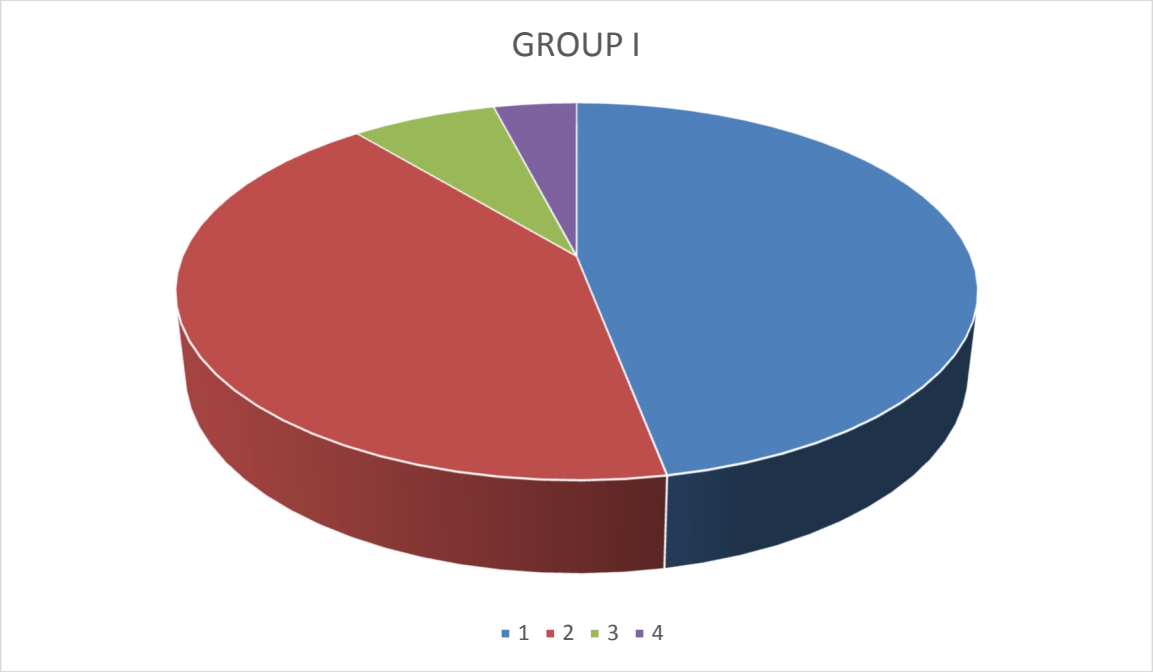
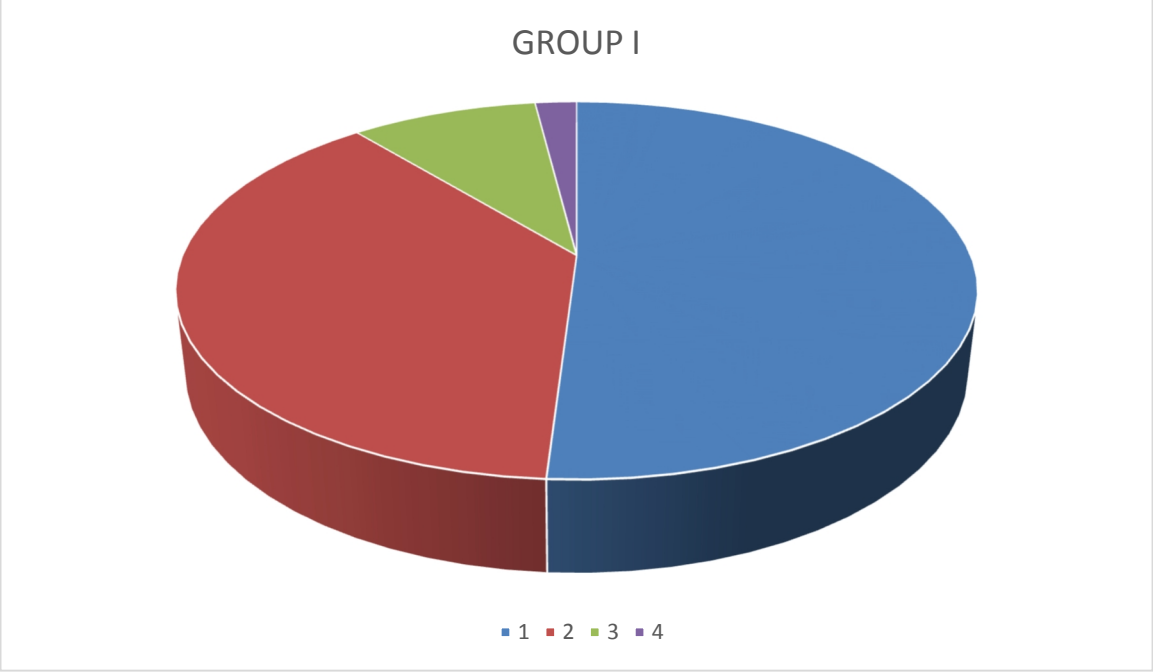


Fig. 3


GROUP I
1
2
3
4


GROUP I
1
2
3
4

Inference

47% of patients in Group I and 51% of patients in Group II were primigravida, while only 4% in Group I and 2% in Group II were Gravida 4.

TABLE - 4

HISTORY OF PIH IN PREVIOUS PREGNANCY

PIH IN PREVIOUS PREGNANCY	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
YES	12	12	5	5
NO	88	88	95	95
Total	100	100	100	100

p = 0.076 ; x2 = 3.150 Not significant

Chi-square = 3.150

HISTORY OF PIH IN PREVIOUS PREGNANCY

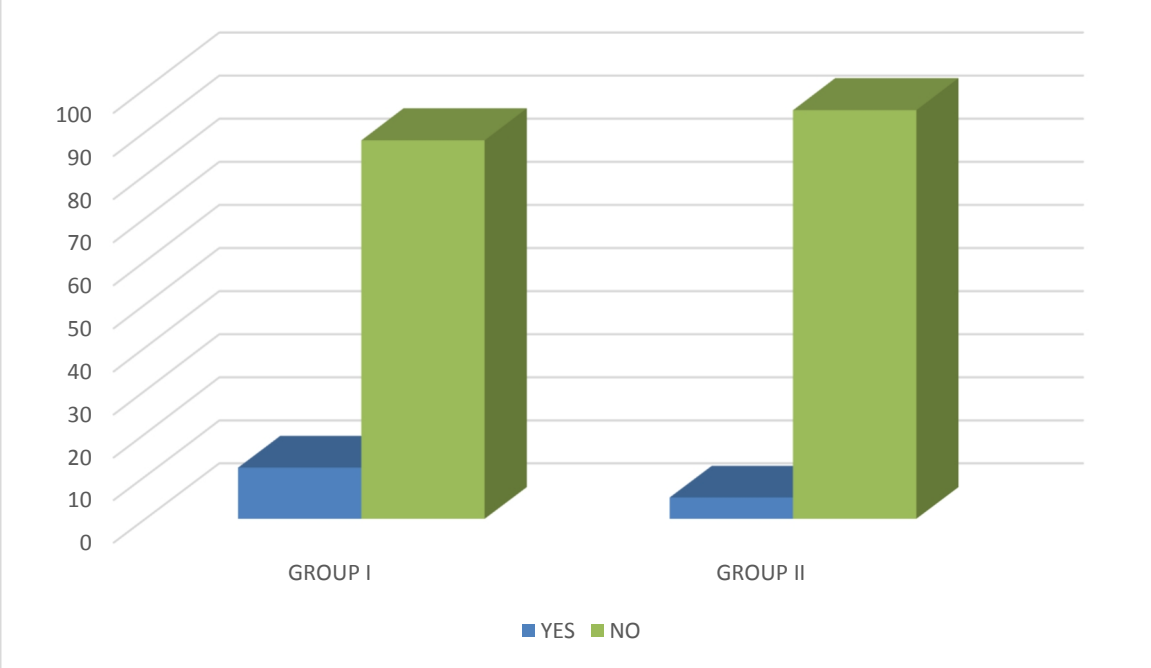
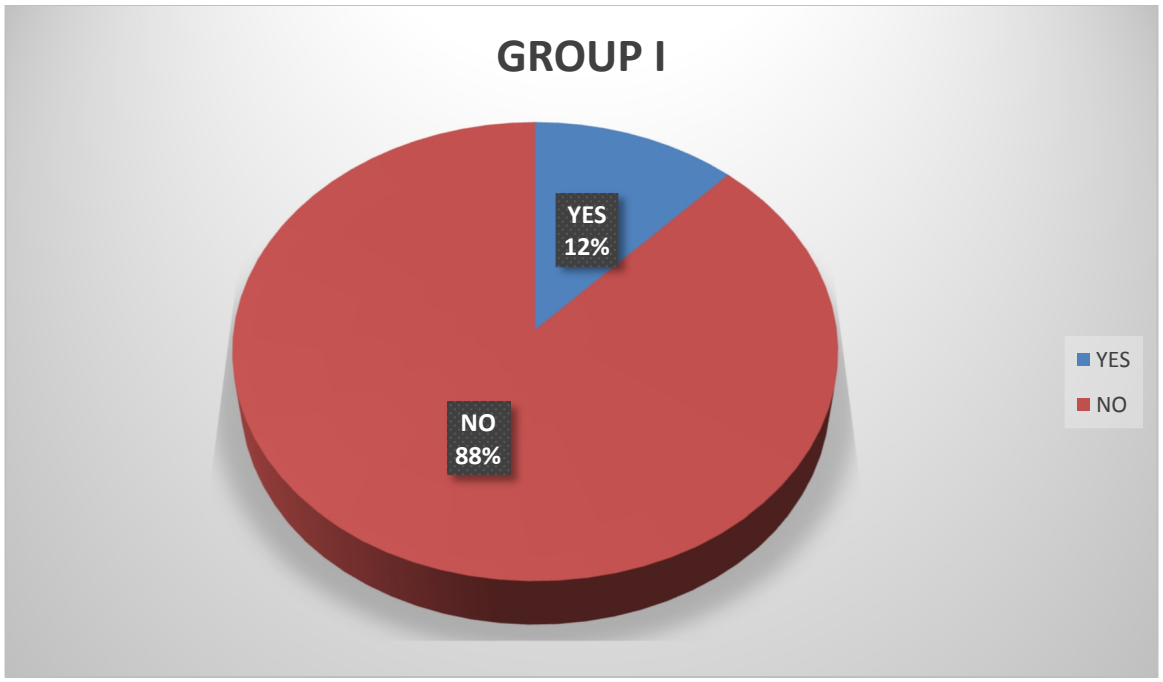
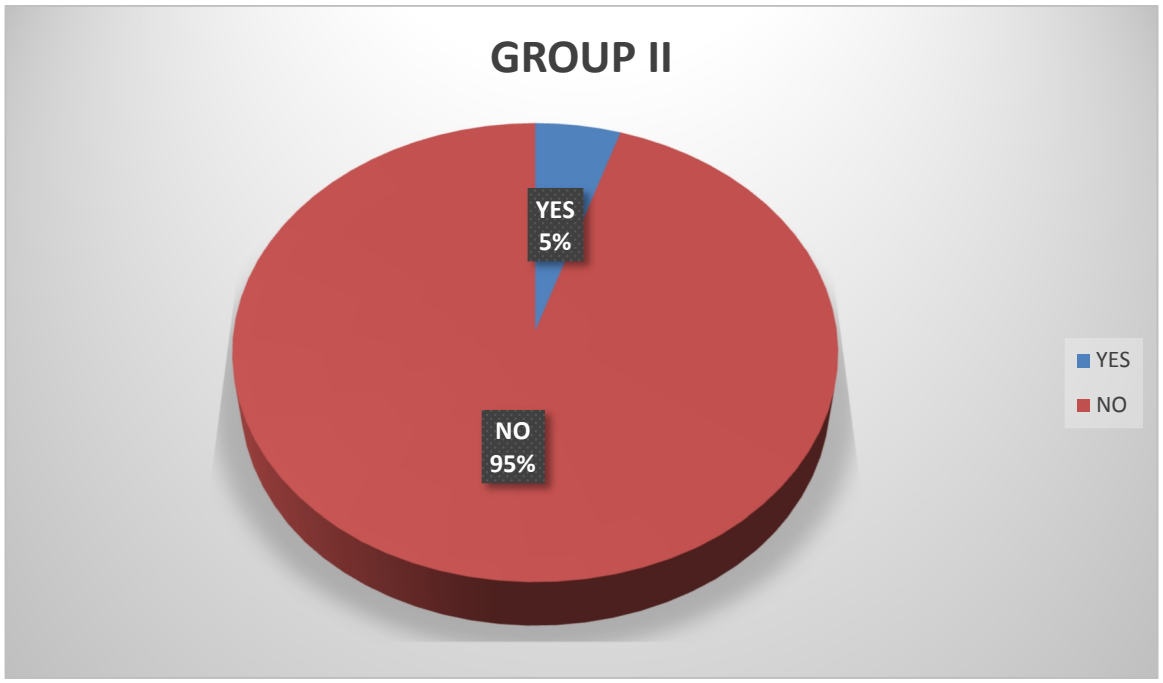


Fig. 4





Inference

12% of patients in group I and 5% of patients in Group II had H/o PIH in the previous pregnancy.

TABLE - 5

COMPARISON OF BMI BETWEEN THE TWO GROUPS

GROUP	NO. OF CASES	MEAN BMI	S.D.	S.E. OF MEAN
GROUP I	100	26.2080	3.39511	0.33951
GROUP II	100	24.710	2.52602	0.25260

p = 0.001 Significant

Inference

It is found that, patients with pre eclampsia(Group I) had high BMI than their normal counterparts (Group II)

TABLE - 6

TOTAL CHOLESTEROL LEVELS

GROUPS	NO. OF CASES	MEAN (mg%)	S.D.	S.E. OF MEAN
GROUP I	100	212.6420	53.10793	5.31079
GROUP II	100	173.7015	41.87940	4.18799

p = 0.03 Significant

Inference

The mean total cholesterol level is significantly higher in pre eclampsia affected mothers in group 1.

TABLE - 7

HDL LEVELS

GROUPS	NO. OF CASES	MEAN (mg%)	S.D.	S.E. OF MEAN
GROUP I	100	41.2684	9.35250	0.93525
GROUP II	100	48.0264	9.71615	0.97162

p = 0.04 Significant

Inference

The mean HDL levels are lower in Group I compared to Group II.

TABLE - 8

VLDL Levels

GROUPS	NO. OF CASES	MEAN (mg %)	S.D.	S.E. OF MEAN
GROUP I	100	48.9432	18.38757	1.83876
GROUP II	100	35.5844	11.18140	1.11814

p = 0.04; T = 6.208 Significant

Inference

The mean VLDL values are significantly higher in Group I compared to Group II.

TABLE - 9

TGL Levels

GROUPS	NO. OF CASES	MEAN	S.D.	S.E. OF MEAN
GROUP I	100	220.4673	62.97049	6.29705
GROUP II	100	169.2040	56.92760	5.69276

$p = 0.035$; $t = 6.039$ Significant

Inference

The mean triglyceride levels show a significant rise in Group I compared to that of Group II.

TABLE - 10

LDL Levels

GROUPS	NO. OF CASES	MEAN (mg%)	S.D.	S.E. OF MEAN
GROUP I	100	135.2441	45.83614	4.58361
GROUP II	100	101.674	38.76126	3.87613

p = 0.045; T = 5.592 Significant

Inference

The mean LDL levels are also increased in Group I compared to that of Group II in a significant manner.

TABLE - 11

MODE OF DELIVERY

MODE OF DELIVERY	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
Vaginal	65	65	69	69
LSCS	35	35	31	31
TOTAL	100	100	100	100

MODE OF DELIVERY

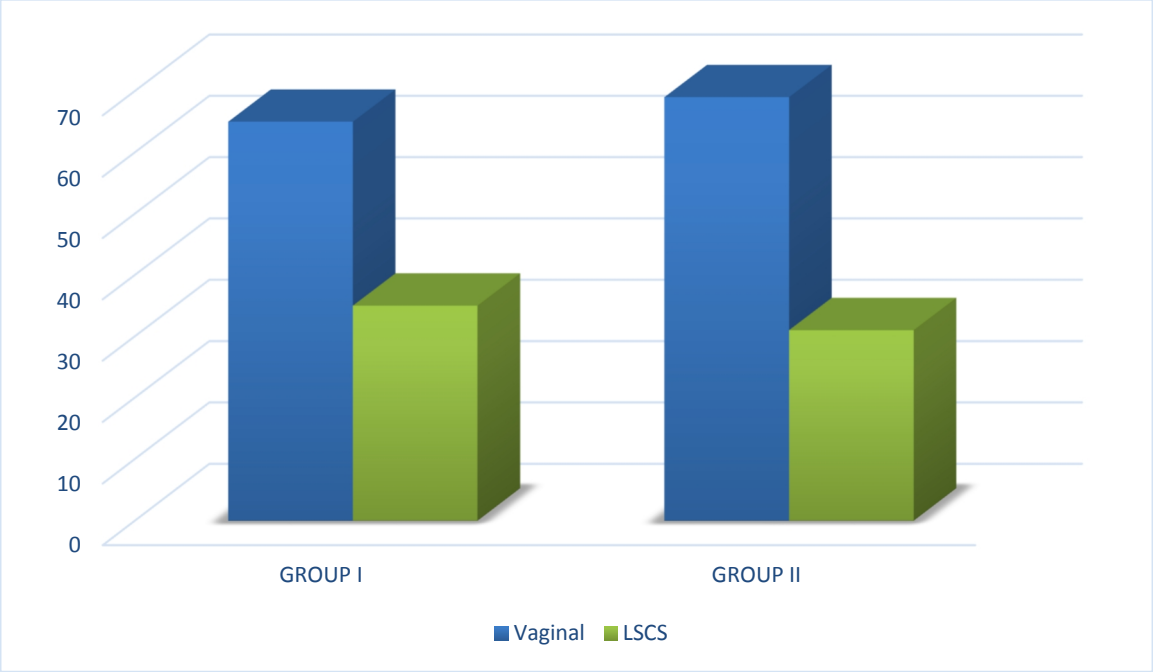
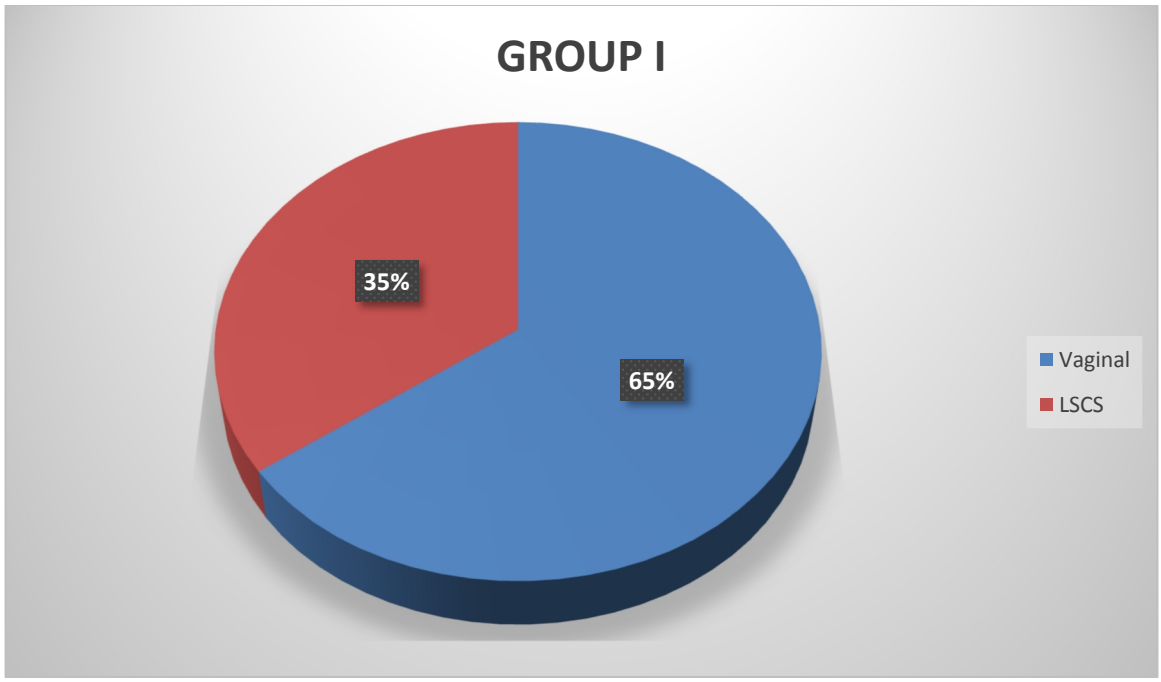
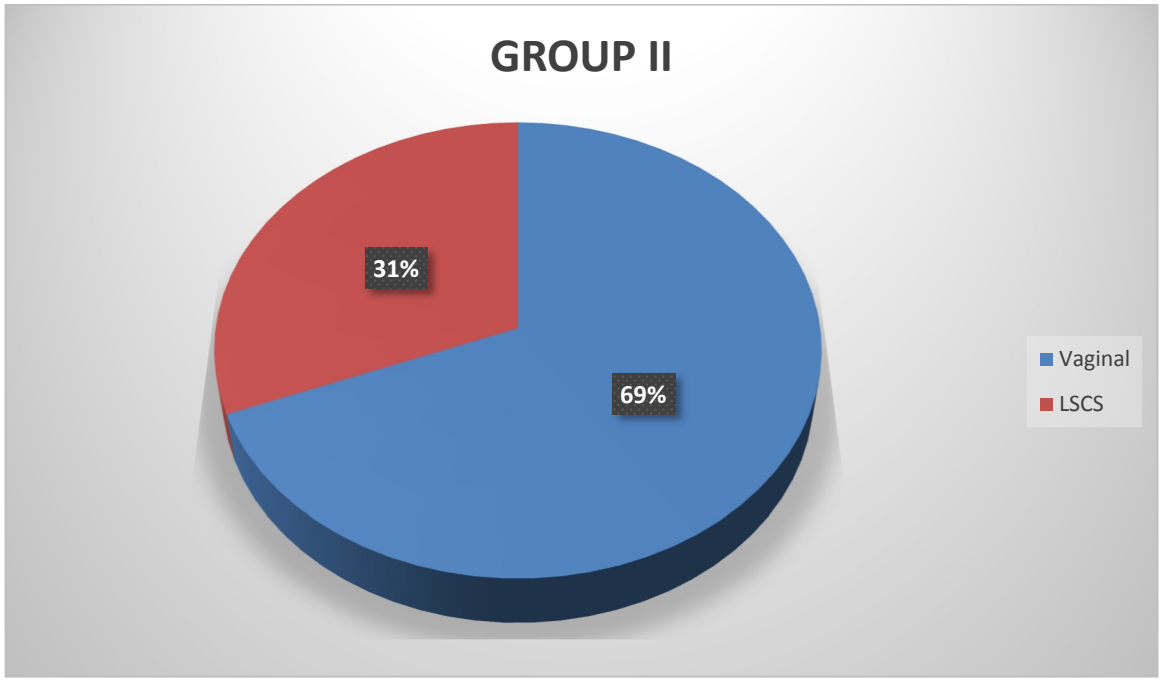


Fig. 5


GROUP I
35%
65%
Vaginal
LSCS


GROUP II
31%
69%
Vaginal
LSCS

Inference

65% of patients in Group I and 69% of patients in Group II had normal vaginal delivery

TABLE - 12

CORRELATION BETWEEN BMI & TGLS

GROUP I

BMI	NO. OF CASES	MEAN OF TGLS (mg%)	S.D.	S.E. OF MEAN
Normal	62	199.3725	53.74081	6.82509
High / OBESE	37	254.0874	63.4071	10.42795

p = 0.02 Significant

GROUP II

BMI	NO. OF CASES	MEAN OF TRIGLYCERIDES (mg %)	S.D.	S.E. OF MEAN
Normal	82	161.6781	51.8977	5.80234
High / OBESE	18	203.5611	69.93061	16.01140

p = 0.025; t = 2.915 Significant

Inference :

It is found that the obese patients or the patients with high BMI in Group I had elevated triglycerides than those with normal BMI in the same group.

Similarly, the patients with high BMI / obese patients in Group II also had elevated triglycerides than the normal.

TABLE - 13

NON OBESE GROUP

GROUPS	NON OBESE	MEAN TGL	S.D.	S.E. OF MEAN
GROUP I	62	127.8315	44.97383	5.71168
GROUP II	81	96.9215	36.13983	4.01554

p = 0.03; t = 4.557 Significant

TABLE - 13 (a) OBESE GROUP

GROUPS	OBESE	EAN TGL (mg %)	S.D.	S.E. OF MEAN
GROUP I	37	254.0874	63.43071	10.42795
GROUP II	18	203.5611	67.93061	16.01140

Variance - 25 - F = 0.676;

Significant = 0.415

2 tailed = 0.009 p = 0.009

Inference :

Among the non obese group, the pre eclampsia patients in Group I had increased triglycerides compared to the normal women in Group II.

Among the obese group also, the mean triglyceride values were higher in patients with pre eclampsia compared to the normotensive pregnant women.

DISCUSSION

In our study, it is found that there is no significant change in age distribution between the two groups.

Our study, similar to that by Ray et al., (2006) shows that the mean triglyceride concentration was significantly higher among the preeclamptic cases than among the unaffected controls.24 What might hypertriglyceridemia predispose a woman to preeclampsia, if there truly exists a causal relationship? A likely factor is the higher risk of placental vasculopathy. Persons with the metabolic syndrome of which hypertriglyceridemia is a major feature, display evidence of chronic inflammation, hypercoagulability and endothelial dysfunction.

Our study similar to Carl Hubel et al., (1995) shows that triglycerides and free fatty acids are increased in preeclampsia.5 Their study correlated the interaction of the lipid peroxidation metabolite, malondialdehyde to endothelial cell dysfunction in preeclampsia. However the effects of malondialdehyde are not included in our study.

The study by Ray et al., (2006) concluded that there exists a correlation between obesity and the risk of preeclampsia.24 Our study also shows that women with elevated BMI in both the control and the PIH

groups had elevated levels of triglycerides and LDL cholesterol. The current study also shows hypertriglyceridemia associated with maternal obesity. Maternal obesity, diabetes mellitus and chronic hypertension, the major features of the metabolic syndrome are positively correlated with the development of preeclampsia in many studies.

The study by Barden, et al., (1999) shows that regardless of parity women with preeclampsia had elevated BMI during pregnancy compared with women who had normotensive pregnancies and triglycerides were significantly elevated in these women and the levels of triglycerides after 6 weeks of delivery were found to the decreased. 2 However in our study, the levels of postpartum triglycerides were not measured.

Our study similar to Sattar et al., (1997) shows the elevated levels of cholesterol and trigycerides.26 Kaaja et al., (1995) has found high plasma triglycerides and low HDL concentrations in women with preeclampsia and gestational hypertension.12 In our study though there is elevated level of cholesterol, and triglycerides, the HDL level is found to be normal.

This study shows that lipid abnormalities can lead to endothelial cell dysfunction and vascular damage, thus playing a role in the pathogenesis of preeclampsia.

The National Heart Foundation of Australia found increased levels of F2 Isoprotane in patients with increased LDL and triglyceride concentrations. Further studies are required in finding the association of F2 Isoprotanes with preeclampsia. They also found significantly reduced HDL levels in preeclamptic patients while in our study the levels of HDL were normal. Further more studies are thus required in determining the association between HDL levels and predisposition to preeclampsia.

Bendomir et al., (1997) studied that lipoprotein subfraction concentrations are higher in preeclampsia.3 In our study, the lipoprotein subfractions were not studied. Further studies are required to find the effect of lipoprotein subfractions in pregnancy.

Anceschi et al., (1992) found increased cholesterol / phospholipid ratio of the erythrocyte membrane in women with pregnancy induced hypertension.1 In our study, the levels of the membrane phospholipids were not measured.

Dempsey et al., (2004) found that women who were small at birth and became overweight in adulthood had an unfavourable lipid profile.7 But the subjects in our study were not aware of their birth weight. Hence this association factor could not be correlated in our study.

Our study similar to that by Kokia et al., (1999) shows significantly elevated levels of triglycerides in the women with pregnancy induced hypertension.15

Thus, the adverse lipid profile found in the hypertensive pregnant patients could be associated with the enhancement of pathological lipid deposition in the predisposed vessels such as uterine arteries.

COMPARISON OF THE LIPID PROFILE ANALYSIS IN VARIOUS STUDIES

	TGLS	LDL	HDL
Ray et al.	↑	-	-
Carl Hubel et al	↑	-	-
Sattar et al	↑	↑	-
Kaaja et al	↑	↑	↓
Our study	↑	↑	↓

SUMMARY

1. There is no significant change in the age distribution between the two groups. 53% of the patients in Group I and 56% of patients in Group II were in the age group of 21 - 25 years. Only 2% of patients in Group I and 4% of patients in Group II were in the age group of 31 - 35 years.
2. The booking status of both the study group and the control group were the same, 92% being booked and 8% being unbooked.
3. 47% of patients in Group I and 51% of patients in Group II were primigravida, while only 4% in Group I and 2% in Group II were Gravida 4.
4. 12% of patients in group I and 5% of patients in Group II had H/o PIH in the previous pregnancy.
5. It is found that, patients with pregnancy induced hypertension (Group I) had high BMI than their normal counterparts (Group II).
6. The mean total cholesterol level is higher in group I than in Group II.
7. The mean HDL levels are higher in Group I compared to Group II.
8. The mean VLDL values are higher in Group I compared to Group II

9. The mean triglyceride levels show a significant rise in Group I compared to that of Group II.
10. The mean LDL levels are also increased in Group I compared to that of Group II. (11)65% of patients in Group I and 69% of patients in Group II had normal vaginal delivery.
11. It is found that the obese patients or the patients with high BMI in Group I had elevated triglycerides than those with normal BMI in the same group. Similarly, the patients with high BMI / Obese patients in Group II also had elevated triglycerides than the normal, in Group II.
12. Among the non obese group, the PIH patients in Group I had increased triglycerides compared to the normal women in Group II. Among the obese group also, the mean triglyceride values were higher in patients with pregnancy induced hypertension compared to the normotensive pregnant women.

CONCLUSION

Analysing and comparing the results between the study group and the control group, it was concluded that :

- 53% of the study group were in the age group of 21 - 25 years.
- 47% of the study group were primigravida.
- BMI was elevated in the Pre eclampsia group.
- Total cholesterol values are significantly increased in pre eclampsia group.
- Triglycerides and LDL were elevated in the Pre eclampsia group, compared to the control group.
- HDL is decreased in pre eclamptic group.
- The obese group with elevated BMI had elevated triglyceride levels.

Hyperestrogenism in pregnancy is responsible for hypertriglyceridemia in pregnancy. Oestrogen induces hepatic bio synthesis of endogenous triglyceride which is arried by VLDL. This increased triglyceride in pre eclampsia is likely to be deposited in predisposed vessels like uterine artery and contributes to endothelial dysfunction directly or indirectly through small dense LDL. Increased

triglyceride and its decreased clearance clearly helps in understanding the pathological process of pre eclampsia. Moreover hypertriglyceridemia is also associated with hypercoagulability.

Thus there exists a consistent positive association between elevated maternal triglyceride and the risk of preeclampsia. Given that the maternal hypertriglyceridemia is a common feature of the metabolic syndrome, interventional studies are needed to determine whether pre - pregnancy weight reduction and dietary modification can lower the risk of preeclampsia.

The matter of whether triglycerides share a causative relationship with preeclampsia should be expanded to the study of other lipoprotein particles and microparticles, as well as a detailed analysis of the microvascular bed of the delivered placenta. The collection of blood specimens in early pregnancy measuring concentrations of insulin, glucose and inflammatory markers, alongside anthropometrics assessment and then followed by a thorough assessment of clinical outcomes through a large cohort study might optimally address the role of triglycerides and the metabolic syndrome in the causation of preeclampsia.

Clearly, there is a need to establish whether preconceptional dietary modification, such as adoption of a Mediterranean diet among obese women, can reduce the future risk of preeclampsia and other placenta mediated diseases, including.

HDL is decreased due to hypo oestrogenic state in pre-eclampsia. Despite hypooestrogenism , insulin resistance in hypertensive disorders favour more towards the reduction in HDL. A tendency to lower serum lipoprotein fractions in women with toxemia and in third trimester of pregnancy is proven in many earlier studies.

It is therefore essential that serum lipid profile should be estimated during routine ante natal care, as it help in predicting pre eclampsia and preventing the major complications of pre-eclampsia before it manifests. Also lipid profile have to be monitored throughout the pregnancy in high risk population as it would be a better predictor of pre eclampsia.

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PROFORMA

Name : Age :

IP No. : Obstetric Score :

Booking Status : Socio Economic Status :

H/o PIH in Previous Pregnancy :

Diet History : Vegetarian

- NonVegetarian

Taking more than two serves per week

Taking less than two serves per

week. Family History :

Religion :

Chief Complaints :

H/o Swelling of legs / oliguria

Any H/o Headache / Blurring of vision / Vomiting / Epigastric pain. Any

H/o DM/HT/Bronchial Asthma /Cardiac/Thyroid Disease. **General**

Examination

Anaemia Jaundice Pedal edema JVP

Vitals:

Any symptoms of imminent eclampsia. Systemic examination:

Respiratory system. Cardiovascular System. CNS:

Obstetric Examination

Investigations:

Albumin

Urine — Sugar

Deposits

24 hrs urinary proteins

VDRL NVP

Blood grouping & typing

CBC - Platelet Count Bleeding time Clotting time

Blood Sugar

Urea

Sr. Creatinine Sr.Uric acid. Sr.Fibrinogen

Liver function Tests

Lipid Profile

Fundus Examination

Obstetric Ultrasound

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

Case										
S.No	**Age**	**Booking status**	**Obstetric Code**	**PIH in previous Pregnancy**	**mean BMI**	**T.Cholesterol**	**HDL Level**	**VLDL Level**	**TGL level**	**LDL level**
1	18	Booked	Primi	No	26.5	250	50	52	250	140
2	22	Booked	G4A3	Yes	27.5	230	52	50	230	141
3	23	Booked	Primi	No	28.4	200	54	55	200	138
4	22	Booked	G2P1L1	No	28.1	172	50	51	172	137
5	24	Booked	G3P2L2	No	26	158	48	49	158	180
6	25	Unbooked	G3P2L2	No	24.1	202	49	48	202	136
7	26	Booked	Primi	No	24	230	50	49	230	134
8	17	Booked	G2P1L1	No	23	235	48	49	235	130
9	28	Booked	Primi	No	18.4	240	49	50	240	121
10	27	Booked	G4P3L3	No	24.1	190	59	56	200	131
11	23	Booked	Primi	No	25.1	182	60	59	182	127
12	24	Booked	G2A1	Yes	28	185	55	56	185	103
13	22	Booked	G2P1L1	No	29.4	192	40	41	192	123
14	19	Booked	Primi	No	21.2	194	41	40	194	124
15	21	Unbooked	G3P2L2	No	22	215	42	42	215	136
16	22	Booked	Primi	No	23.4	232	45	43	282	141
17	23	Booked	G2P1L1	No	25	280	44	44	230	142
18	24	Booked	Primi	No	26.1	149	48	49	149	146
19	25	Booked	G2P1L1	No	27.4	189	49	48	189	151
20	32	Booked	Primi	No	28.9	212	50	50	212	136
21	21	Unbooked	G4P3L3	No	27.6	220	52	51	220	129
22	24	Booked	Primi	No	26.9	232	52	53	232	131
23	26	Booked	G2A1	Yes	28	210	52	53	210	135
24	28	Booked	G3P2L2	No	26.8	194	54	54	194	136
25	29	Booked	G2P1L1	No	28.4	190	56	55	190	170

26	30	Booked	G2P1L0	No	30.5	184	48	49	184	121
27	25	Booked	Primi	No	26.1	192	42	40	192	136
28	21	Booked	G2P1L1	Yes	27	199	48	47	199	139
29	24	Booked	G2P1L1	No	26	168	46	47	168	143
30	23	Booked	Primi	No	19.6	152	40	41	152	147
31	24	Booked	G2P1L1	No	34.5	190	41	42	190	118
32	30	Booked	Primi	No	25	285	40	40	205	129
33	26	Booked	Primi	No	26.5	224	50	49	224	129
34	28	Booked	G2P1L1	No	27.5	240	52	50	240	120
35	22	Booked	Primi	No	28.4	234	54	54	234	147
36	23	Booked	G2P1L1	No	28.1	239	50	50	239	143
37	24	Booked	Primi	No	26	280	48	49	280	139
38	21	Booked	G2A1	Yes	24.1	294	49	50	294	136
39	22	Booked	Primi	No	24.5	280	50	49	280	121
40	27	Booked	Primi	No	24	192	48	49	192	170
41	27	Booked	G2P1L1	No	23.5	173	49	48	73	136
42	28	Booked	G2P1L1	No	18.4	293	55	56	293	135
43	24	Unbooked	G2P1L1	No	24.1	218	60	59	218	131
44	25	Booked	Primi	No	25.1	249	55	54	249	129
45	21	Booked	Primi	No	28	322	40	41	322	136
46	22	Booked	G2P1L1	No	29.4	289	41	39	289	135
47	23	Booked	Primi	No	21.2	194	42	38	194	131
48	29	Booked	G2P1L1	Yes	22.5	184	45	37	184	129
49	28	Booked	Primi	No	23.4	281	44	45	281	136
50	24	Booked	G2P1L1	No	28.5	294	48	48	294	151
51	29	Booked	G4P3L3	No	26.1	280	49	48	280	146
52	25	Unbooked	Primi	No	29.4	270	50	52	270	142
53	24	Booked	G2P1L1	No	28.9	182	52	53	182	141
54	28	Booked	Primi	No	29.6	184	52	54	184	146

55	21	Booked	Primi	No	26.9	156	54	55	156	124
56	26	Booked	G2P1L1	No	28	190	52	58	190	123
57	24	Booked	G2P1L1	Yes	26.8	148	56	45	148	103
58	27	Booked	Primi	No	28.4	194	48	49	194	127
59	25	Booked	Primi	No	30.5	190	42	40	190	131
60	29	Booked	Primi	No	28.1	185	48	46	185	121
61	21	Unbooked	G2P1L1	No	27.5	180	46	49	180	130
62	28	Booked	G2P1L1	Yes	28.5	170	40	41	170	134
63	22	Booked	Primi	No	19.6	180	41	42	180	136
64	29	Booked	G2A1	No	34	185	40	40	185	180
65	21	Booked	Primi	No	25.5	190	48	39	190	140
66	30	Booked	Primi	No	27.3	210	50	52	285	129
67	29	Booked	G2P1L1	No	28.4	218	52	54	190	120
68	30	Booked	Primi	No	25.1	224	54	57	152	147
69	21	Booked	G2P1L1	No	26.5	240	50	50	168	143
70	26	Booked	Primi	No	27.5	234	48	47	199	139
71	22	Booked	Primi	No	28.4	239	49	48	192	136
72	28	Booked	G2P1L1	No	28.1	280	50	51	184	121
73	27	Unbooked	G3P2L2	No	28	294	48	52	190	170
74	25	Booked	G2P1L1	No	24.1	280	49	49	194	136
75	30	Booked	G3P2L2	No	24.5	192	55	60	260	139
76	25	Booked	G2A1	Yes	23.5	173	60	51	322	131
77	27	Booked	Primi	No	18.4	293	55	54	220	129
78	28	Booked	Primi	No	24.1	218	40	40	212	136
79	25	Booked	G2A1	No	25.1	249	41	49	189	139
80	29	Booked	G2P1L1	No	28	322	42	43	240	131
81	24	Booked	Primi	Yes	29.9	289	49	45	230	129
82	26	Booked	G2P1L1	No	21.2	184	44	48	282	136
83	27	Booked	Primi	No	24.5	184	48	49	215	151

84	21	Booked	Primi	No	23.4	281	49	51	294	146
85	28	Booked	G2P1L1	No	25	284	50	54	192	142
86	29	Booked	G2P1L1	No	26.1	280	52	55	185	141
87	21	Unbooked	Primi	No	27.4	260	52	51	182	136
88	30	Booked	G2A1	Yes	28.9	182	52	59	260	124
89	22	Booked	G3P2L2	No	29.6	180	54	48	230	123
90	27	Booked	Primi	No	26.9	154	56	59	236	103
91	28	Booked	Primi	No	28	190	48	48	230	127
92	24	Booked	Primi	No	26.8	148	42	47	202	131
93	22	Booked	G2P1L1	No	28.4	194	48	40	158	121
94	25	Booked	Primi	No	30.5	190	46	41	172	130
95	34	Booked	G2P1L1	No	26.1	185	40	48	200	134
96	22	Booked	G2A1	No	27	180	41	47	230	136
97	23	Booked	Primi	No	28	170	40	42	250	180
98	21	Booked	G2P1L1	Yes	19.6	180	48	41	212	140
99	30	Booked	Primi	No	34	185	47	44	221	141
100	26	Booked	Primi	No	25.5	190	48	49	220	137

CONTROL										
S.No	**Age**	**Booking status**	**Obstetric Code**	**PIH in previous Pregnancy**	**mean BMI**	**T.Cholesterol**	**HDL Level**	**VLDL Level**	**TGL level**	**LDL level**
1	18	Booked	Primi	No	26.4	138	40	32	134	101
2	32	Booked	Primi	No	25	152	38	33	148	102
3	23	Booked	Primi	No	24.1	146	37	32	142	98
4	21	Unbooked	G4P3L3	No	23	160	29	34	156	96
5	22	Booked	G2P1L2	No	24.2	162	42	33	158	99
6	28	Booked	Primi	Yes	26.2	184	33	35	180	96
7	16	Booked	G2P1L1	No	27	180	34	36	186	91
8	31	Booked	Primi	No	24.2	172	36	37	168	93
9	23	Booked	Primi	No	24.6	200	37	35	196	121
10	29	Booked	G2P1L1	No	27	190	38	34	186	118
11	24	Booked	Primi	No	35	180	39	38	176	108
12	19	Booked	Primi	No	28	176	46	35	174	108
13	25	Booked	G2P1L1	No	23	148	47	37	144	107
14	21	Booked	G2P1L1	No	21.1	156	46	40	152	105
15	30	Booked	Primi	No	28	180	38	41	176	101
16	22	Unbooked	G4P3L3	No	27	180	39	33	172	99
17	23	Booked	Primi	No	25.5	170	38	34	160	91
18	26	Booked	G2P1L1	No	26	168	49	35	161	90
19	24	Booked	Primi	No	27	162	50	39	153	88
20	27	Booked	Primi	No	21	152	39	40	140	101
21	19	Booked	Primi	No	20	140	36	41	150	104
22	33	Booked	G2P1L1	No	18.5	152	47	30	151	94
23	25	Booked	Primi	No	24	160	48	29	170	94
24	28	Booked	G2P1L1	Yes	25.5	173	47	33	169	98
25	29	Booked	Primi	No	26	172	46	35	184	99

26	21	Booked	Primi	No	27.5	188	38	40	190	98
27	23	Booked	Primi	No	23	192	49	43	188	120
28	29	Unbooked	G3P2L2	No	21.5	190	50	41	180	124
29	22	Booked	G2P1L1	No	20	188	47	35	190	108
30	24	Booked	Primi	No	20.5	190	48	36	218	105
31	25	Booked	Primi	No	19	228	49	37	224	104
32	30	Booked	Primi	No	20.8	234	50	40	173	98
33	26	Booked	Primi	No	26.4	138	58	39	178	96
34	23	Booked	Primi	No	29	152	40	32	178	96
35	22	Booked	G2P1L1	No	30	146	38	33	173	98
36	21	Booked	G2A1	No	24	160	37	32	220	104
37	26	Booked	G2P1L1	No	23	162	29	34	212	105
38	24	Booked	G3P2L2	No	24.2	184	42	33	188	108
39	25	Booked	Primi	No	26.2	190	33	35	186	124
40	22	Unbooked	G2P1L1	Yes	27	172	34	36	188	120
41	27	Booked	Primi	No	24.2	200	36	37	186	98
42	21	Booked	Primi	No	24.6	190	37	35	184	99
43	24	Booked	G2P1L1	No	27	180	38	34	169	98
44	23	Booked	Primi	No	35	176	39	33	170	94
45	25	Booked	G3P2L2	No	28	148	46	35	151	94
46	28	Booked	Primi	No	23	156	47	37	150	104
47	22	Booked	G2P1L1	No	21.1	180	46	40	140	101
48	21	Booked	Primi	No	28	180	38	41	153	88
49	23	Booked	Primi	No	27	170	39	33	161	90
50	29	Booked	G2P1L1	No	25.5	168	38	34	160	91
51	24	Booked	G2P1L1	No	26	162	49	35	171	99
52	27	Booked	Primi	No	27	152	50	39	176	101
53	25	Unbooked	Primi	No	21	140	39	40	152	105
54	30	Booked	G3P2L2	No	20	152	36	41	144	107

55	22	Booked	Primi	No	18.5	160	37	30	174	108
56	23	Booked	Primi	No	24	173	48	29	176	118
57	21	Booked	Primi	No	25.5	172	47	33	186	108
58	26	Booked	G2A1	No	26	188	46	35	196	121
59	24	Booked	Primi	No	27.5	192	48	40	168	93
60	25	Booked	Primi	No	23	190	49	43	184	91
61	22	Booked	G3P2L2	No	21.5	188	50	41	180	96
62	27	Booked	G2P1L0	Yes	20	190	47	35	158	99
63	21	Booked	Primi	No	20.5	228	48	36	156	96
64	30	Booked	Primi	No	19	234	49	37	142	98
65	28	Booked	G2P1L1	No	20.8	173	50	40	148	102
66	29	Booked	G3P2L2	No	20.8	138	50	39	134	101
67	29	Booked	Primi	No	19	152	40	32	134	101
68	25	Booked	Primi	No	20.5	146	38	33	148	102
69	23	Booked	Primi	No	20	160	37	32	142	98
70	30	Booked	Primi	No	21.5	162	29	34	156	96
71	21	Booked	G2P1L1	No	23	184	42	33	158	99
72	26	Booked	G2P1L1	No	27.5	180	33	35	180	96
73	22	Booked	Primi	No	26	172	34	36	186	91
74	26	Unbooked	G3P2L2	No	25.5	200	36	37	168	93
75	24	Booked	Primi	No	24	190	37	35	196	121
76	23	Booked	G2P1L1	No	18.5	180	38	34	186	118
77	27	Booked	G2P1L1	No	20	176	39	33	170	108
78	25	Booked	Primi	No	21	148	46	35	172	107
79	28	Booked	G2P1L1	No	27	156	47	37	132	108
80	21	Booked	G2P1L1	No	26	180	46	40	152	105
81	28	Booked	G2P1L1	No	25.5	180	38	41	176	101
82	22	Booked	G2P1L1	No	27	170	39	33	172	99
83	23	Booked	Primi	No	28	168	38	34	160	91

84	29	Unbooked	G3P2L2	Yes	21.1	166	40	35	161	90
85	24	Booked	Primi	No	23	152	50	39	151	98
86	25	Booked	G2P1L1	No	28	140	39	40	139	101
87	30	Booked	G2P1L1	No	35	152	36	41	150	104
88	21	Booked	G2P1L1	No	27	160	47	30	151	94
89	26	Booked	G2P1L1	No	24.6	173	48	29	170	94
90	22	Booked	Primi	No	24.2	172	47	33	165	98
91	27	Booked	G2P1L1	No	27	188	45	35	184	99
92	23	Booked	G2P1L0	No	26.2	192	38	40	188	120
93	28	Booked	Primi	No	24.2	190	49	43	180	124
94	17	Booked	G2A1	No	23	188	30	41	190	108
95	29	Booked	G2A1	No	24	190	47	35	218	105
96	24	Booked	G2P1L1	No	25	228	48	36	224	104
97	30	Unbooked	G3P2L2	No	26.4	234	49	37	173	98
98	25	Booked	G2P1L1	No	27	173	38	40	169	96
99	34	Booked	Primi	No	30	174	39	39	168	99
100	18	Booked	Primi	No	29	174	40	30	167	105